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Table of Contents.

THE RH BLOOD TYPES IN AUSTRALIAN ABORIGINES.

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As an introduction to this paper, which presents our findings of the Rh blood types and probable Rh gene frequencies in pure-blooded Australian aborigines, it may be of interest to restate some of the views expressed by leading anthropologists concerning the physical characteristics, possible origin and racial mixture of this unique race.

Howells (1937), in reporting his analysis of anthropometric data collected by W. L. Warner in Arnhem Land and on Bathurst and Melville Islands, discussed at some length the Australian race problem. His views and those advanced by others prior to that date were ably presented in that paper and full references were given. We therefore intend to quote briefly the theories summarized by Howells and to proceed further with views expressed since 1937 by other authors.

Comparisons with Other Racial Groups.

The origin of the Australians has always been one of the most absorbing topics of human phylogeny. Here in Australia is found the most primitive existing physical type combined with the most primitive material culture, yet these people have been able to formulate and administer the most formidable systems of kinship known to anthropology.

What, then, are their relationships with the rest of Homo sapiens? Several authors to the contrary, there is little reason to suppose that the physical type we recognize as Australian evolved in Australia, or that it has always been confined to Australia. There is an analogy in the American Indian; nobody now claims that he evolved in the New World; however, the facts in this case are more obvious. As with the Indian, one must search out the Australian's remote past by looking among other groups for people who may be related to him. There are suggestions that an "Australoid type" was once an important element in the populations of other parts of the world, even in America; the calvarium from Punin, Ecuador, is a case in point. Our task is therefore to find indications of the former range of the Australian type, before any estimate can be made as to the time and extent of its greatest importance.

As a preface to comparisons, the physical type of the Australian must be recapitulated. Of its external features the combination of a very black skin with hair that is not frizzly, but wavy or curly, is the most noteworthy. The long, narrow, keeled cranium with its beetling brows is another characteristic complex. The mouth region, with its long lips, projecting alveolar processes, large palate, and receding chin, is another. The nose is very short and bulbous, with wide and thick alæ. The cumulative effect argues strongly for the theory that the Australian is a survivor from an actual level in the morphological development of man, and not merely a peripheral subracial type.

Metrical characters are a very dolichocephalic head and a nose which on the living is as broad as it is long. The face, though not extreme, is moderately broad and short.

Discussion of the Australian Race.

Theories. Prester John and the Ten Lost Tribes have hardly given rise to opinions more multifarious and romantic than has disputation over the ultimate origin of the Australians. And small wonder, for, as we have said, they are peculiar people, aberrant from the three major race groups, and confined to one corner of the earth. Their flowing wavy hair has always been the chief actual cause for argument, but equally arresting facts are the above-mentioned isolation and their undeniably primitive morphology.

The following are some of the theories which have been offered by various anthropologists, and which were discussed in turn by Howells:

I. Australia represents the home of mankind.
II. The Australians are descended from Neanderthal man.
III. They are the living representatives of an early Homo sapiens stage.
IV. They are the product of mixture between: (*a*) a "white" and a Negrito or Negro stock, (*b*) two differing Negroid strains, (*c*) Tasmanian and Polynesian.

Of the theories presented, Howells prefers the third and goes on to make the following statement:

The Australians an Early Form of Homo Sapiens.

The theory to which we are led by the present body of evidence is more or less the one which Keith suggested. The Australian is not a blend but a major race, and is the most archaic race still surviving. He probably presents with considerable fidelity the morphological stage attained by Homo sapiens in Asia at that remote time when he wandered out into the Pacific and isolation. He may have undergone some minor changes, but he is intrinsically what he was when he left the evolutionary crucible on the Asiatic mainland which has since given off the specialized (superficially) Negroid and Mongoloid and the unspecialized white and American Indian.

Howells then summarizes his views as follows:

1. The Australians, characterized by a uniformly primitive morphology throughout the head and cranium, are, considering the area over which they are extended, remarkably homogeneous in physical type. Admixtures of Melanesian blood, which must have taken place in the north, have had little effect.
2. The Australians are a major race which represents an earlier stage in the development of Homo sapiens than does any other existing race. It has been preserved, with its primitive features, through stagnation and isolation; its characteristic wavy hair is probably older than the woolly hair of the Negro.
3. Representatives of the Australoid race, mixed with other strains, are to be found in Tasmania, New Caledonia, the Bismarck Archipelago and Ceylon, and possibly also in Southern India and some of the Lesser Sunda Islands. The Sakai and Toala, however, are not related to the type, being rather of Negrito derivation.
4. The Australoid type originated on the Asiatic continent, perhaps in India, and spread into the Western Pacific as the first representative of modern man, probably at a very remote period. Outside of Australia, the Australoids have been extinguished or submerged everywhere except in a few marginal regions.

Other Views.

Griffith Taylor (1937 and 1945), basing his views on anthropometric, ethnological and geographical data, considers the basic Australian with his wavy hair to be ethnically higher than the Papuan (Negro) with his woolly hair, who in turn represents a later evolutionary stage than the Negrito. According to his migration-zone theory, successively evolved races migrating from a more or less common centre of evolution, which he places in Central Asia, have forced out their predecessors into more remote or peripheral areas. Thus the Australian, evolved later than the Negro and Negrito, migrating probably under pressure from a later evolved race, left Asia later than did the Negro and Negrito. To explain the absence of Negroid characteristics in Australia, he (1945) suggests that the following may have taken place:

Perhaps we may assume a Negrito migration during an early glacial period, when it was easy to get into Papua and also into Melanesia and Australia. Then the Papuan hordes moved to the south after Australia was shut off by the drowning of the Sunda region. They entered Papua at a time when we may imagine extensive deserts in northern Australia for reasons given earlier. They drove the Tapiro into the hill-jungles. Later the first horde of Australians arrived during an Ice Age, when the Sunda and Sahul areas were dry land. They moved down the central rivers to the south-east. They mixed with the Negritoes in the south to some extent, producing the Tasmanian type. The next migration drove the Tasmanian further south, and was in turn driven into the thick forested regions of New South Wales by the latest migration.

Taylor thus differs from Howells in his belief that the Australian is not the most primitive racial type extant, and that he does not represent a single stage in the morphological development of man, but rather is the result of admixture of several distinct migrations.

Birdsell (1947, personal communication) has most kindly given us an outline of his unpublished work and has generously permitted us to publish a brief summary of his views on this interesting race.

My work was done in the so-called marginal areas never previously covered in any detail, extending from Perth around the south and east up to Cairns. The populations from these areas clearly show, on a geographical basis of analysis (not typing), that *three* major racial groups contributed to the composition of the present aborigines.

(*a*) Negritic: The Oceanic Negritos were the first to reach Australia and have left their genotypic impact on some of the marginal populations living today. This is most evident in the Barrinians of the rain-forest region in the Cairns area, and among the now extinct Tasmanians. However, other marginal groups show some evidence of Negritic admixture, especially in the extreme south-west of Western Australia, and in the coastal plains of the east protected by the Great Dividing Range. Traces are also present among the Tiwi of Melville and Bathurst Islands. It should be emphasized that nowhere today in Australia are "pure" Negritos found, but there is ample evidence for their presence in mixed form.

(*b*) Murrayian: This is an ancient basic white race, closely allied to the Ainu of Japan. It is present in predominant form, roughly, in the southern half of Australia. It is found least mixed in Murray drainage (hence the name) and the coastal regions to the south and east. It is light skinned, large headed and faced, rather short and stocky in build, with excessive body and facial hair, and generally "white" appearance morphologically.

(*c*) Carpentarian: This tall, linear dark race, like the other two, may be traced back to the Asiatic mainland, where its closest affinities are with the Vedda of Ceylon and the Munda of India. This is the racial element which is certainly basic in all the very dark skinned peoples of India. In Australia, it is predominant in the northern half of the continent (in a rough way). It represents the last major people to populate the country (excluding, of course, casual Malay and Torres Straits infiltrations on the north coast, and white colonization). Their date of arrival, via the Sahul Shelf, may be placed at about the end of the Pleistocene or the beginning of the recent period.

As you can see, this picture differs very substantially from that presented by Howells and others, but I have every reason to believe that the data I have in hand now supports this interpretation and in fact demands it.

From the foregoing it is apparent that the precise position of the Australian aborigines in human phylogeny has not been satisfactorily determined, although painstaking scientific investigations have provided a sound basis of knowledge on which plausible constructive theories have been built up.

The examination of blood grouping data for confirmation or otherwise of these theories makes an interesting study. Prior to 1940 a number of blood grouping investigations on Australian aborigines had been made by various workers (Tebbutt and McConnel, Cleland *et alii*, Lee, and Phillips). From their reports it appeared that the B factor was lacking in the Australian except in the north, where it was considered to be of recent introduction. These findings placed the aborigines in a position with regard to blood groups as unique as that which they occupied with regard to morphological characteristics, and indicated a lack of affinity with existing predominant Asiatic groups. A resemblance was apparent between the blood grouping pictures of the American Indian and the Australian.

The accumulated data of these earlier workers, which covered the O-A-B groups only, have been listed with complete references by Boyd (1939).

During 1938-1939 a field survey in Australia was carried out by J. B. Birdsell on behalf of the Harvard University and the University of Adelaide. This investigation covered both a detailed anthropometric analysis and a study of

the blood groups and M,N types on 805 and 730 aborigines respectively.. The survey extended from Western Australia to Queensland; but the majority of the aborigines tested were in these two States. The results and interpretations of the blood grouping work were published in an excellent paper by Birdsell and Boyd (1940).

Just prior to the publication of the paper by Birdsell and Boyd, an investigation had been commenced at these laboratories on Australian aborigines, and this survey dealt with the blood groups, subgroups of A and the M,N types. In all, 703 aborigines were tested and the blood samples were obtained from Port Hedland, Broome, Derby, Wyndham, Darwin, Melville and Bathurst Islands, Alice Springs, Oodnadatta, Menindee, Cherbourg, Woorabinda and Groote Eylandt.

These two surveys were largely complementary in that they covered different areas. The results of the latter investigation (Wilson, Graydon, Simmons and Bryce, 1944) enabled the informative yet hypothetical "isogenes" of Birdsell and Boyd to be extended to cover practically the whole of the continent.

When tests for the Rh factor in blood became possible, samples from 281 aborigines who originated in Queensland, New South Wales, South Australia and the Northern Territory were tested in this laboratory. All were found to be Rh-positive (Simmons, Graydon, and Hamilton, 1944). Subsequently, further series of blood samples have been collected from various states and the Rh types determined. The results obtained form the basis of this communication.

Materials and Methods.

This investigation was made possible by the generous cooperation of our named collaborators and others.

The samples, consisting of two drops of blood, were collected into small bottles of sterile Rous and Turner glucose-citrate preserving solution (Simmons and Graydon, 1945), and were returned to us by post, or as air freight from the more distant localities. The samples were not packed in ice, as there was little delay in transit.

The anti-Rh testing sera used were anti-Rh^{o} (anti-D), anti-Rh′ (anti-C), anti-Rh″ (anti-E), and anti-Hr′ (anti-c). The three anti-Rh sera were found locally and were pure samples of their particular type. The anti-Hr′ serum used was a sample of the original serum found by Dr. Peter Vogel, and our small sample came to us from Dr. A. S. Wiener, of New York. This potent antiserum was used in preliminary tests at a dilution of 1 in 20, and as a check serum at a dilution of 1 in 7.

While this investigation was in progress it was suggested to one of us (R.T.S.) by Dr. J. B. Birdsell, of the Peabody Museum, United States of America, that we obtain blood samples from the last of the living "Murrayians" who were known to number 33 in 1939. The selection was based on the detailed genealogical investigations carried out in 1939 by Norman B. Tindale, of the South Australian Museum. At that time these aborigines were living at the Point McLeay and Lake Tyers aboriginal stations in South Australia and Victoria respectively. In 1947 it was found that some of these natives were widely scattered, and that death had reduced their number to nineteen. All the survivors were successfully traced, and blood samples were obtained from eleven of those who were prepared to cooperate. We are very grateful to all those police officers and local doctors who traced a number of these aborigines and collected the blood samples for us. Tests covering the blood groups, subgroups of A, M,N types and Rh subtypes were carried out on these samples, and the results have been sent to Dr. Birdsell. These "Murrayian" Australians will be the subject of further comment in a comprehensive paper to be published by Birdsell in the United States of America at a later date. The Rh typing results on these aborigines are shown in Table I; they are grouped together under Lake Tyers and Point McLeay localities.

The nomenclature for the Rh phenotypes used in this paper is that suggested by Snyder (1945). This nomenclature has been used in our series of papers to date dealing with blood anthropology, and is used again on the grounds of consistency, and to avoid possible confusion. The reactions of the antigens corresponding to the Rh phenotypes and genotypes with the various Rh and Hr antisera, together with several Rh nomenclatures suggested (including that of the present writers), have been set out in tabular form by Graydon and Simmons (1946).

Results.

As in the previous investigation, all the aborigines tested (234) were found to be Rh-positive. A total of 515 aborigines have now been Rh tested without the discovery of one who was Rh-negative. (The term "Rh-positive" is used throughout this paper in its broad sense to include samples reacting with any of the three types of anti-Rh sera.)

In Table I are shown the Rh types as determined for aborigines in various Australian States on samplings which may be considered to be representative of the aborigines today. The specimens, which were collected for us by

TABLE I.

Distribution of the Rh Types and Subtypes in Australian Aborigines (Determined by the Use of Anti-Rh^{o}, Anti-Rh′, Anti-Rh″ and Anti-Hr′ Testing Sera).

Locality.	Collector of Specimens.	Number of Subjects Tested.	rh	Rh$^{o'}$		Rh$^{o''}$	Rh$^{o'}$Rh$^{o''}$		Rh^{o}	Rh′		Rh″	Rh′Rh″
				Rh$^{o'}$Rh$^{o''}$	Rh$^{o''}$Rh^{o}		Rh$^{o'}$Rh$^{o''}$	Rh$^{o''}$Rh^{z}		Rh′Rh′	Rh′rh		
			− − −+	+ + − −	+ + − +	+ − + +	+ + + +	+ + + −	+ − − +	− + − −	− + − +	− − + +	− + + +
Oodnadatta, South Australia.	Mounted Constable A. C. Collins ..	48	0	16	8	11	12	0	1	0	0	0	0
Woorabinda, Queensland.	Mr. C. Jensen ..	63	0	27	9	4	17	4	1	1	0	0	0
Yarrabah, Queensland.	Rev. R. S. Campbell	55	0	39	4	1	7	0	1	3	0	0	0
Alice Springs, Northern Territory.	Dr. L. C. Lum ..	23	0	10	0	2	10	1	0	0	0	0	0
Lake Tyers, Victoria Point Mcleay, South Australia.	Mr. T. Milliken Mr. C. Bartlett and others.	11	0	5	0	1	5	0	0	0	0	0	0
Roper River, Northern Territory.	Dr. John Heath ..	34	0	15	3	1	13	2	0	0	0	0	0
Totals ..	.. .. ..	234	0 (0)	112 (47·9%)	24 (10·3%)	20 (8·5%)	64 (27·4%)	7 (3·0%)	3 (1·3%)	4 (1·7%)	0	0	0

field collaborators, were taken from aborigines who had originated in widely scattered districts, and were with few exceptions from adults.

The Rh typing results are summarized in Table I, from which the following frequencies of occurrence of the "basic" Rh phenotypes may be obtained: $Rh^{o\prime}$, 58·2%; $Rh^{o\prime\prime}$, 8·5%; $Rh^{o\prime}Rh^{o\prime\prime}$, 30·4%; Rh^{o}, 1·3%; Rh', 1·7%. No examples of phenotypes rh (Rh-negative), Rh″ or Rh′Rh″ were found.

The use of the anti-Hr′ testing serum enabled the basic phenotypes $Rh^{o\prime}$, $Rh^{o\prime}Rh^{o\prime\prime}$ and Rh′ to be further subdivided as shown in the table. Most of the Rh phenotypes (Table I), as determined by the use of four testing antisera, may consist of two or more genotypes; for example, phenotype $Rh^{o\prime}Rh^{o\prime}$ may contain genotypes $R^{o\prime}R^{o\prime}$ and $R^{o\prime}R'$; phenotype $Rh^{o\prime}Rh^{o\prime\prime}$ may contain as many as nine different genotypes if all the Rh genes are represented.

There are three exceptions, phenotypes Rh′Rh′, Rh′rh and rh, the genotypes of which are unequivocally determined by the use of the four antisera.

It has been the custom to designate each phenotype by the name of the most common included genotype; thus the phenotype giving "++−+" reactions respectively with the three anti-Rh sera and anti-Hr′ serum is generally called $Rh^{o\prime}$rh, because $R^{o\prime}r$ is the common genotype in this phenotype among the white races. However, the gene *r* has not been found in the Australian aborigines, so that the use of the title $Rh^{o\prime}$rh would be misleading if applied to that phenotype in the Australian. The only genotype giving "++−+" reactions in this race is $R^{o\prime}R^{o}$, and we have therefore used the designation $Rh^{o\prime}Rh^{o}$ for the corresponding phenotype. In other cases in which one of the eight basic phenotypes has been subdivided, the principle of naming the subdivisions according to their most common included genotype has been followed. Though convenient in the present instance, such names are unsatisfactory in tables comparing phenotype frequencies occurring in different races. Until a more adequate system of naming the phenotypes has been developed, it would seem preferable to continue the common laboratory practice of using as headings signs indicating the reactions with the test sera in a standard order; for example, the sign "++−+" would head the column which includes all phenotypes whose reactions with anti-Rh^{o}, anti-Rh′, anti-Rh″ and anti-Hr′ sera are "positive", "positive", "negative" and "positive" respectively. Such signs have been included in Table I. In a later table of this paper (Table IV) only the eight basic phenotypes, separable by the use of the three anti-Rh sera, have been listed. Consequently the signs given represent the reaction with the three anti-Rh sera only.

TABLE II.

Rh Types Observed, Compared with Numbers Expected from the Calculated Gene Frequencies.

Phenotype.	Number Observed.	Number Expected.
$Rh^{o\prime}Rh^{o\prime}$	112	108·47
$Rh^{o\prime}Rh^{o}$	24	27·69
$Rh^{o\prime}$	20	17·47
$Rh^{o\prime}Rh^{o\prime\prime}$	64	67·93
$Rh^{o\prime}Rh^{z}$	7	6·85
Rh^{o}	3	1·71
Rh'	4	3·88

The Rh Gene Frequencies.

The gene frequencies and their standard errors, which have been kindly calculated for us by Mr. E. J. Williams, of the Section of Mathematical Statistics, Council for Scientific and Industrial Research, Melbourne, according to the method of maximum likelihood (Fisher, 1946) are as follows: $R^{o\prime} = 0{\cdot}5642 \pm 0{\cdot}0367$; $R^{o\prime\prime} = 0{\cdot}2009 \pm 0{\cdot}0193$; $R' = 0{\cdot}1287 \pm 0{\cdot}0324$; $R^{o} = 0{\cdot}0854 \pm 0{\cdot}0140$; $R^{z} = 0{\cdot}0208 \pm 0{\cdot}0079$. In Table II the number to be expected within each phenotype in a sample of 234 individuals drawn from a population having the above gene frequencies is compared with that actually found.

The agreement between observed and expected values is good ($\chi^2 = 1{\cdot}56$ for one degree of freedom), so that if there is any heterogeneity in the population sampled it is not revealed in the pooled data.

The percentage of positive reactions found with anti-Rh′ serum was 90·2, and with anti-Hr′ serum 47·4, and when these results were subjected to the statistical tests as described by Graydon and Simmons (1946), it was found that *D* for the Rh′ − Hr′ system was 0·038 with a standard error (σ_D) of 0·033. The value 1·2 for D/σ_D suggests that technical errors have not significantly affected the results.

Source of the Rare Subtypes Rh^{o}, Rh′Rh′ and $Rh^{o\prime}Rh^{z}$ in Australia.

While the actual source (district) of the rare subtypes Rh^{o}, Rh′Rh′ and $Rh^{o\prime}Rh^{z}$ found in this series is not of direct importance in the analysis and discussion presented in this paper, it is thought that the details which we have should be recorded, as they may assume greater importance when these results are further analysed from the anthropological point of view. In Table III are recorded the source, State, tribe and district of origin of the natives possessing the rare Rh subtypes.

TABLE III.

Source of Rare Rh Subtypes in Australian Aborigines.

Rh Subtype.	Source.	State.	Tribe.	District of Origin.
Rh^{o}	Oodnadatta.	South Australia.	Not stated.	Not stated.
	Woorabinda.	Queensland.	Not stated.	Bedford.
	Yarrabah.	Queensland.	Laura.	North Queensland.
Rh′Rh′	Woorabinda.	Queensland.	Not stated.	McIvor.
	Yarrabah.	Queensland.	Cooktown.	North Queensland.
	Yarrabah.	Queensland.	Mossman.	North Queensland.
	Yarrabah.	Queensland.	Cooktown.	North Queensland.
$Rh^{o\prime}Rh^{z}$	Woorabinda.	Queensland.	Not stated.	Yandanburra.
	Woorabinda.	Queensland.	Not stated.	Gayndah.
	Woorabinda.	Queensland.	Not stated.	Roma.
	Woorabinda.	Queensland.	Not stated.	Rockhampton.
	Alice Springs.	Northern Territory.	Arunta.	Central Australia.

Additional Blood Group and M,N Data Obtained on Australian Aborigines.

During the progress of this investigation, some aborigines were tested whose blood groups and M,N data have not been reported previously.

At Alice Springs, Dr. L. C. Lum collected 23 blood samples from 16 natives of the Arunta tribe and seven from the Mount Doreen tribe. Of these, eleven were of group O, twelve were of group A_1, and four were of type M, eight of type MN and eleven of type N.

From the aborigines of the Roper River area on the fringe of Arnhem Land a number of samples were collected by Dr. John Heath. No blood grouping data had been reported previously for this area. Of the 34 samples tested, 23 were of group O, eight of group A, two of group B and one of group AB. There were four samples of type M, thirteen of type MN and seventeen of type N. These figures, though admittedly small, suggest the "recent" introduction of the B factor extended into Arnhem Land. The M,N results ($m = 0{\cdot}31$, $n = 0{\cdot}69$) agree well with those reported for neighbouring areas by Wilson *et alii* (1944).

Discussion.

Rh Types in Australian Aborigines Compared with Other Races.

In Table IV the Rh types found in various races are presented. The table is arranged firstly in ascending order of Rh-negative percentages, and then partly in order of type $Rh^{o\prime}$ percentages. On reading down, it is interesting to note the grading of the results, in that, as the type $Rh^{o\prime}$ percentage decreases, type $Rh^{o\prime\prime}$ appears and then pro-

gressively increases in percentage, as also does type Rh°′Rh°″. In a number of races tested to date only three major types, Rh°′, Rh°″ and Rh°′Rh°″, have been detected; this applies particularly to Papuans, Admiralty Islanders, Fijians, Filipinos, Maoris and Japanese.

In Indonesians type Rh° appears as 0·5% and type Rh′Rh″ as 0·5%. The percentage for type Rh′Rh″ is, however, not a true indication, as this type has actually been detected only once, or possibly twice, in approximately 600 tests, and is therefore extremely rare. Of the 600 Indonesian samples tested, 304 have been Rh typed and 296 tested with anti-Rh° serum and with a polyvalent anti-Rh° and anti-Rh′ serum. Additional Rh typing details on one lot of 104 Javanese will appear in a later paper.

In American Indians (100% Rh-positive) both types Rh° and Rh′ have been detected. In Chinese type Rh° has been detected. In Asiatic Indians both types Rh° and Rh′ have been found. The American Negroes, not necessarily pure blooded, have shown both types Rh° (over 40%) and Rh′ (about 2·0%). Recent figures obtained by the French for African Negroes show an even higher percentage for type Rh° (Mourant, 1947, personal communication).

In the present series of Australian aborigines, both types Rh° and Rh′ have been detected.

Table IV, which shows the Rh types only, does not indicate the races in which the rare gene Rh^z (or Rh^y) has been detected. They are as follows: (i) whites (English), Murray *et alii* (1945); (ii) whites (Australians), Simmons *et alii* (1945); (iii) whites (Americans), Wiener *et alii* (1946); (iv) Mexican Indians, Wiener *et alii* (1945, 1946); (v) Papuans, Simmons *et alii* (1946); (vi) Indonesians, Simmons and Graydon (1947).

The Presence of the Rare Rh Subtypes in Australian Aborigines in Relation to Fisher's Crossover Theory.

Consideration of the relative frequencies of the Rh genes in the large English series reported by Fisher and Race (1946) led those authors to suggest that the rarer Rh "genes" might have resulted from the crossing over of the "gene-elements" in the more common heterozygotes. The three common heterozygotes occurring in their series—$R^{o\prime}r$, $R^{o\prime}R^{o\prime\prime}$ and $R^{o\prime\prime}r$—would produce by crossing over R^o and R', R^o and R^z, and R^o and R'', respectively. If this mechanism was responsible for the maintenance of the observed frequencies, one would expect that the frequency of "gene" R^o would be approximately equal to the sum of the frequencies of R', R'' and R^z. This was the case in the English series.

In the present survey the common heterozygotes are $R^{o\prime}R^{o\prime\prime}$, $R^{o\prime}R'$, $R^{o\prime}R^o$, $R^{o\prime\prime}R'$, and $R^{o\prime\prime}R^o$. Three of these are heterozygous with regard to one factor only and could not produce new genes by crossover. The triple heterozygote $R^{o\prime\prime}R'$, which represents only about 5% of the population, could produce three pairs of genes on crossover, $R^{o\prime}$ and R'', R^o and R^y, and R^z and r. It is noticeable that one gene of each of these pairs was not found in the series, so that it seems most unlikely that crossover from the heterozygote $R^{o\prime\prime}R'$ could have been a significant factor in producing the observed gene frequencies. The remaining and most common heterozygote, $R^{o\prime}R^{o\prime\prime}$, would produce by crossover "genes" R^o and R^z in approximately equal quantities. Both of these were observed in the survey; but their frequencies, 0·085 and 0·021 respectively, differed more than would be expected were crossing over responsible for their maintenance. The relatively high frequency of gene R' cannot be explained on the basis of the crossover theory. In any population the gene R' could be produced by crossover in six different ways, three from doubly heterozygous genotypes and three from triple heterozygotes. None of these heterozygotes has been shown to exist in the Australian aborigines, so another explanation must be sought for the occurrence of gene R'.

In a personal communication (1947) Dr. R. R. Race suggested that the gene element D^u which determines a rather labile antigen D^u might be present in this series, and that the anti-Rh° (anti-D) serum used may have failed to react with certain cells possessing weakened D^u antigen. It is impossible to test this suggestion adequately without repeating the series, but attempts are being made to obtain fresh samples from the four subjects who were classified as Rh′Rh′. It should be pointed out, however, that the anti-Rh° serum used in this survey possessed anti-D and anti-D^u activity and was found to give very definite reactions with D^u cells kindly sent to us from England by Dr. P. Renton, of the Manchester Blood Transfusion Service.

It may be relevant to state here that the reactions are considered to be indicative of the presence of the R^z gene; but without familial investigations we have no means of distinguishing in such cases between the genes R^z and R^y. Gene R^z has been preferred because it has been definitely proved to exist in the English (Murray, Race and Taylor, 1945) and because it might be produced by crossover in the most commonly occurring heterozygote $R^{o\prime}R^{o\prime\prime}$.

The gene R^y predicted by Fisher has not yet been demonstrated. Basing his calculations on the observed frequencies

TABLE IV.

Distribution of the Rh Blood Types (as Determined by the Use of Three Sera: Anti-Rh°, Anti-Rh′, Anti-Rh″).

Population.	Authors.	Number of Subjects Tested.	(Rh+)[1]	Percentage of Rh Types.							
				(Rh−)rh	Rh°′	Rh°″	Rh°′Rh°″	Rh°	Rh′	Rh″	Rh′Rh″
				−−−	++−	+−+	+++	+−−	−+−	−−+	−++
Papuans.	Simmons *et alii* (1946)	100	100	0	93·0	0	7·0	0	0	0	0
Admiralty Islanders.	Simmons and Graydon (1947)	112	100	0	92·9	0·9	6·2	0	0	0	0
Fijians.	Simmons and Graydon (1947)	110	100	0	89·1	1·8	9·1	0	0	0	0
Filipinos.	Simmons and Graydon (1945)	100	100	0	87·0	2·0	11·0	0	0	0	0
Indonesians.	Simmons and Graydon (1947)	200	100	0	74·0	2·5	22·5	0·5	0	0	0·5
Australian Aborigines.	Simmons *et alii* (present survey)	234	100	0	58·2	8·5	30·4	1·3	1·7	0	0
American Indians—											
Mexico.	Wiener *et alii* (1945)	95	100	0	48·1	9·5	41·2	1·1	0	0	0
Oklahoma.	Wiener *et alii* (1946)	105	100	0	40·0	17·1	39·1	2·9	0·9	0	0
Maoris.	Simmons *et alii* (incomplete)	32	100	0	25·0	31·0	41·0	3·0	0	0	0
Japanese.	Miller and Taguchi	180	99·4	0·6	51·7	8·3	39·4	0	0	0	0
Japanese.	Waller and Levine	150	98·7	1·3	37·4	13·3	47·3	0	0	0	0·7
Chinese.	Wiener *et alii* (1944)	132	98·5	1·5	60·6	3·0	34·1	0·9	0	0	0
Asiatic Indians (Moslems).	Wiener *et alii* (1945)	156	92·9	7·1	70·5	5·1	12·8	1·9	2·6	0	0
American Negroes.	Wiener *et alii* (1944)	223	91·9	8·1	20·2	22·4	5·4	41·2	2·7	0	0
American Negroes.	Levine (1945)	135	92·6	7·4	23·7	16·3	4·4	45·9	1·5	0·7	0
Puerto Ricans.	Torregosa (1945)	179	89·9	10·1	39·1	19·6	14·0	15·1	1·7	0·5	0
White, Americans.	Wiener *et alii* (1946)	766	87·5	12·5	54·7	14·9	14·0	2·2	0·9	0·5	0
White, Americans.	Unger *et alii* (1946)	7317	85·3	14·7	53·5	15·0	12·9	2·2	1·1	0·6	0·03
White, English.	Fisher and Race (1946)	927	85·2	14·8	54·9	12·2	13·7	2·5	0·7	1·3	0
White, English.	Murray (1946)	1038	84·7	15·3	54·8	14·7	11·6	2·3	0·6	0·7	0
White, Australians.	Simmons *et alii* (1945)	350	85·1	14·9	54·0	12·6	16·6	0·6	0·9	0·6	0
White, Hollanders.	Graydon *et alii* (1946)	200	84·6	15·4	51·5	12·3	17·7	1·5	1·5	0	0
White, French.	Bessis (1946)	501	83·0	17·0	51·7	13·6	13·0	3·6	0·4	0·8	0

[1] In this table Rh+ includes all samples reacting with anti-Rh°, anti-Rh′ or anti-Rh″ sera.

in the above-mentioned English series, Fisher has deduced that, according to his crossover hypothesis, the frequency of R^r is of the order of 48 per million. If the phenotype frequencies in other races given in Table IV are inspected for heterozygotes capable of producing the gene R^r by crossover, the impression is gained that the incidence of that gene is unlikely to be appreciable in any part of the world. Of course, this surmise would have to be revised if some mechanism other than crossover (such as abnormally high mutation rates) was shown to be responsible for the maintenance of the existing frequencies of Rh genes.

When examined in the light of the brief review introducing this paper, Table IV reveals the inadequacy of our knowledge of the Rh types in the ethnic components of the races of the South-West Pacific Area. Figures are not available for any of the racial groups mentioned as having affinities with the Australian aborigines. The American Indian whose A B O blood grouping picture resembles that of the Australian has Rh type frequencies remarkably similar also. Only in respect of the M,N type frequencies are they sharply differentiated. These findings suggest a more or less contemporary existence in Asia of the progenitors of these two races, at a time when a north-south gradient of the gene *m*, as envisaged by Birdsell and Boyd (1940), was present. If this was so, it would support Taylor's theory that the Australian represents a later evolutionary stage than the Negro.

The Melanesian groups have a much higher incidence of type $Rh^{o'}$ than the aboriginal, and a correspondingly lower incidence of type $Rh^{o''}$. These differences and the presence of group B in the Melanesian clearly distinguish between the two races. The Rh type frequencies of the Australian aboriginal are closer to those of the so-called "higher" races than are those of the Melanesians. Incidentally, the Rh type frequencies found in the latter bear no resemblance to those reported for the American Negroes, which adds further to the dissimilarity between the blood-grouping pictures of these two negroid types.

No knowledge of the Rh or M,N types is available for Negritoes in any part of the world, nor have we blood grouping data for the aborigines of the rain forest area west of Cairns, in which group Negrito affinities were found by Birdsell. A comprehensive blood-grouping survey on this Australian group and on Negrito populations, particularly those existing as "inliers" on the migration route from Asia, should yield valuable evidence on the Negrito influence in Australia. There are several such groups of Negritoes, significantly, in the most inaccessible places; but it is hoped to obtain a representative sampling from one such group in New Guinea in the near future.

The affinity of the Murrayian and the Ainu might also be reexamined with profit. In neither the A B O groups nor the M,N types of the Ainu and the southern aboriginal can any resemblance be seen. It is unfortunate that a more representative sampling of the true Murrayian is not available today. The determination of the Rh types existing in the Ainu would throw further light on this point.

Even less is known of the blood-grouping picture of groups outside Australia that are believed to be related to the Carpentarian. Again there are a few "inliers" on the migration route, but the main representatives are the Vedda of Ceylon and the Munda of India. Inquiries made through Dr. R. L. Spittel, of Colombo, have revealed that it would now be practically impossible to obtain even a small sampling of pure-blooded Vedda, as few are known to exist today in Ceylon. However, it may still be possible to obtain satisfactory data for the Munda. It is noted that the pre-Dravidians so far examined (Boyd, 1939) have had, for India, an abnormally low frequency of *q*, the gene for the B factor, which agrees with the thesis that they are proto-Australian. The presence of the B factor could be accounted for by slight recent admixture with the neighbouring Indian populace, which has the highest frequency of *q* in the world.

It would seem appropriate here to sound a note of caution regarding the inferences to be made from Rh type frequencies in racial studies. The Rh factor is the only blood-grouping antigen known to have a survival value. It can be shown that this survival advantage tends to eliminate the less common gene from a population. We have no knowledge of any compensatory mechanisms by which the existing frequencies might be stabilized. This suggests that in addition to chance fluctuations, the incidence of the Rh types is continually changing under directional influences. It would appear, then, that the Rh type frequencies existing in the various races today relate to a past perhaps less remote than do the data for the other known blood factors.

The incompleteness of the data provides the spur to stimulate further racial studies in the South-West Pacific Area, which perhaps ethnologically is the most interesting in the world. Attempts will be made to fill in some of the gaps indicated in our knowledge by obtaining blood samples from natives of the central mountain area of New Guinea, from the Negritoes of the Nassau Mountains in Dutch New Guinea, from the Munda of India and from the Ainu of Japan, and generally to extend our Pacific studies as opportunity arises.

Summary.

1. The Rh types of 234 pure-blooded Australian aborigines have been determined. The percentages found were as follows: $Rh^{o}Rh^{o'}$, 47·9; $Rh^{o}R^{o}$, 10·3; $Rh^{o''}$, 8·5; $Rh^{o}Rh^{o''}$, 27·4; $Rh^{o}Rh^{z}$, 3·0; Rh^{o}, 1·3; $Rh'Rh'$, 1·7. The Rh gene frequencies calculated from these results were as follows: $R^{o'}$, 0·564; $R^{o''}$, 0·201; R', 0·129; R^{o}, 0·085; R^{z}, 0·021.

2. A total of 515 aborigines have been tested for the Rh factor and all have proved to be Rh-positive.

3. Some additional blood grouping and M,N typing data for natives of Central Australia and the Roper River district, Northern Territory, are reported.

4. No evidence in confirmation of Fisher's crossover theory could be seen in the calculated gene frequencies.

5. Various theories advanced by anthropologists relating to the Australian race problem have been presented and to some extent discussed. Consideration of these theories reveals the incompleteness of the blood-grouping data for the South-West Pacific Area. Plans for further Pacific studies are outlined.

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ANEURYSMS OF THE SPLENIC ARTERY, WITH A REPORT OF TWO CASES AND REVIEW OF THE LITERATURE.

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ANEURYSMS of the splenic artery are of sufficient rarity to be always worthy of recording. Crisp[1] is quoted by Sherlock as having published the first report in 1847, and since then approximately 145 cases have been recorded in the literature. In this paper a report is made of two additional cases. Sherlock and Learmonth[2] found 0·039% of splenic artery aneurysms in their survey of over 84,000 autopsies. Lennie and Sheehan[3] give the figure as 0·05% for aneurysms of the splenic artery and 1·26% for the incidence of all types of aneurysms.

There appears to be an interesting relationship between the occurrence of these aneurysms and the sex and age of the patients as well as pregnancy. Of Sherlock and Learmonth's series the sex of 98 patients was known, and of these, 60 were females and 38 males. The average age was about forty-eight years in each of the two sexes. Before the age of forty-five years there is a striking sex difference, Lennie and Sheehan stating the ratio of females to males as 34:12. In contrast, they give the occurrence of aneurysms of the renal artery for the under forty-five years age group as 21 males to 7 females in their series.

Of the 60 females mentioned in Sherlock and Learmonth's series, 15 were pregnant, and Lennie states that half the aneurysms which occurred in women aged from fifteen to forty-five years were associated with pregnancy. There does not appear to be sufficient evidence to decide whether pregnancy plays a part in the causation of aneurysms of the splenic artery.

Ætiology.

Aneurysms of the splenic artery have been attributed to many causes, and amongst the more common are arterial degeneration, syphilis, congenital defects of the vessel wall, splenomegaly, endocarditis, trauma and pregnancy. Of the less commonly postulated causes, increase of pressure in the portal circulation, invasion from a gastric carcinoma, thrombosis and arterio-venous communication have been mentioned. According to Allen, Barker and Hines,[4] necrotizing arteritis and periarteritis nodosa may be the cause. The more important ætiological factors will be dealt with more fully.

Arterial Degeneration.

Most of the aneurysms of the splenic artery found incidentally *post mortem* are probably due to arteriosclerosis, and hypertension, when present, is probably an important factor in their enlargement. Danforth[5] states that calcification of the arterial wall with attendant weakening is an important factor in their occurrence. The sac of an arteriosclerotic aneurysm is thin and calcified in patches and contains laminated clots. Generally there is evidence of atherosclerosis elsewhere.

Syphilis.

Syphilis is probably a very rare cause, and of Sherlock and Learmonth's series of 125, in only two, in their opinion, was there conclusive evidence of this ætiological factor. Binder, quoted by Danforth, states that syphilis plays no part except in the case of aneurysms of the aorta.

Congenital Defects of the Vessel Wall.

In most cases of splenic aneurysm due to defects of the vessel wall, there will be evidence of similar defects elsewhere in the arterial tree, and aneurysms of the circle of Willis are a not uncommon concomitant finding. There may be a defect in or atrophy of the media and internal elastic lamina at the point of division of the main artery or of its larger branches. Histological examination may reveal such defects; but according to Lennie, microscopic examination gives no clue to the ætiology in the vast majority of cases.

Splenomegaly.

Lennie quotes Beneke, who suggested that the spleen and its artery should be considered as a single unit, and that increase in the size of one component would give rise to an increase in the size of the other. Attempts at hypertrophy of the splenic artery might be followed by premature degenerative changes in its walls. However, there seems to be little evidence to support this view.

Endocarditis.

Danforth quotes Ponfick, who suggests that endocarditis is important ætiologically, and believes that an embolus at the point at which an artery branches in loose supporting tissue may be the determining factor which produces aneurysm. In patients with mycotic aneurysms, there was usually evidence of rheumatic valvulitis, and multiple splenic infarcts were noted in seven of the twelve cases of aneurysms of the splenic artery attributed to this cause in the series reviewed by Cosgrove *et alii*.[6]

Trauma.

The anatomical position of the splenic artery and its proximity to important viscera make the chance of survival after injury to the splenic vessels unlikely.

Pregnancy.

It is striking that in Sherlock and Learmonth's series of splenic aneurysms, pregnant women form 20% of the number of females and 11% of all their patients. The relationship is probably more apparent than real, however, and it seems likely that these aneurysms would have remained quiescent but for the changes in abdominal pressure accompanying pregnancy. These pressure changes probably caused symptoms or precipitated rupture of the aneurysms without playing any part in their ætiology. This explanation is the more probable, since the preponderance of cases associated with pregnancy came to notice in the later months of pregnancy or just after delivery.

Pathological Anatomy.

Sherlock and Learmonth in their series found that the aneurysms were single in 74% and multiple in 26%. They quote Ware (1856) as having reported a patient with ten aneurysms in the splenic artery. In 51% of the series the main artery was affected, in 40% a branch, and in 8% both the main trunk and its branches. Degenerative aneurysms most commonly arose from the main vessel, those due to congenital defects at points of division.

Clinical Features.

There may be no signs or symptoms, and aneurysms may be found incidentally. Even when rupture of an aneurysm of the splenic artery is the immediate cause of death, there may be nothing to suggest it in the patient's history. The symptoms and signs will obviously vary according to whether the patients are examined before or after the rupture.

From a review of the literature, Cosgrove states that upper abdominal pain, vomiting and indigestion are not uncommon symptoms before rupture, whilst some patients present themselves complaining of anorexia or weakness, loss of weight or even constipation. Some, as the first patient in the present paper, may merely have noticed a lump in the abdomen, or, as in the second case, the symptoms, if any, may be masked by some concurrent disease. Of the signs found in patients examined before rupture has occurred, enlargement of the spleen and a systolic bruit over a palpable mass are not uncommon findings.

FIGURE I.
Photograph of the two aneurysms of the splenic artery in Case I.

Sperling[7] suggests the following triad as being helpful in the diagnosis: (*a*) left upper quadrant pain, (*b*) a systolic bruit over a palpable tumour mass, and (*c*) a pulsating filling defect in the greater curvature of the stomach revealed radiologically. Seids and Hauser[8] have reviewed the radiological diagnosis of these aneurysms, and mention circular or oval opacities with calcified outlines as being helpful diagnosic aids. Lower and Farrell[9] suggest examination of the stools in suspected cases for undigested fat, in order to detect a lessened pancreatic activity which may be due to the pressure of the mass on the pancreas.

It would seem that before rupture the differential diagnosis must include gastric and duodenal ulceration, gastric carcinoma, chronic pancreatitis, cholelithiasis and hydatid or mesenteric cyst. Even after rupture the clinical picture is not clear cut, as both Gillam[10] and Parsons[11] stress the fact that rupture of an aneurysm of the splenic artery may be a primary or a two-stage process.

Sherlock and Learmonth give the order of frequency of the cavities into which hæmorrhage occurred by single or primary rupture in 56 cases as follows: (i) the peritoneum via the lesser sac (34); (ii) the stomach (12); (iii) retroperitoneal tissues (5); (iv) small intestine (4); (v) colon (4); (vi) pancreas (4); (vii) the splenic vein (1). Murphy[12] gives an account of an aneurysm of the splenic artery eroding the stomach wall and causing death from hæmatemesis in a man aged sixty-six years. Most of these patients presented with an acute abdominal condition, with sudden epigastric pain, muscular rigidity, hæmatemesis or melena.

Of the "two stage" rupture, Gillam states that the initial hæmorrhage into the lesser sac may not be fatal, and if it is recognized, appropriate treatment by splenectomy gives a good chance of recovery. In most of the cases recorded in the literature in which the "two stage" rupture occurred, the secondary rupture took place about two weeks after the primary rupture and proved fatal. The latent period, however, varied in some cases from a few hours to eight weeks. In the second case report, the secondary rupture occurred four weeks after the primary event. Gillam records a case in which such a "two stage" rupture of an aneurysm of the splenic artery occurred in a woman during pregnancy.

The differential diagnosis of a ruptured splenic aneurysm must, of course, include all the causes of an acute condition of the abdomen. A ruptured ectopic pregnancy or concealed accidental hæmorrhage may be falsely diagnosed, especially owing to the not infrequent association of these aneurysms with pregnancy, and in some cases the epigastric pain may even suggest preeclampsia. The condition is undoubtedly rare, but its possibility should be borne in mind. Danforth states that enough cases have occurred in association with pregnancy to cause the obstetrician to keep in mind the possibility, if not the probability, of such a condition when upper abdominal pain occurs, especially if it is accompanied by the signs of loss of blood. Sherlock and Learmonth found that the clinical diagnosis had been made in only ten of the 125 cases they reviewed.

Prognosis.

Even in the absence of pregnancy, the outlook is very poor. An indication of this poor prognosis is given by Machemer and Fuge.[13] Of their collected series, in 25 cases in which no surgical treatment was attempted, there were 18 deaths. In 13 cases in which surgical treatment was tried there were seven deaths. In some of these cases the treatment was merely tamponade. In eight cases in which the splenic artery was tied there were two deaths. In all the cases so far reported as occurring in association with pregnancy, only two patients have survived.[10][14]

Treatment.

Scrutiny of the procedures used in the series reviewed suggests that before rupture the procedure of choice, if possible, is splenectomy with removal of the segment of the splenic artery from which the aneurysm has arisen.

After rupture the only method of dealing with the catastrophe is rapid recognition of the condition, heroic surgery and blood transfusion. The affected artery must be ligated proximal to the aneurysm, and if possible the spleen must be removed. Lennie and Sheehan state that if the condition occurs in association with pregnancy, the surgeon must, after having excluded any obstetric cause for the hæmorrhage, perform a rapid Cæsarean section to clear the field. They consider that the fœtus need not be considered, as it is always dead.

Reports of Cases.

CASE I.—Miss C.F., aged forty-two years, reported to hospital for examination during May, 1947. She stated that she had noticed a lump in the upper part of her abdomen on the left side for the past twelve months. This lump was not associated with any pain or subjective symptoms. Her appetite was good, she experienced no discomfort after meals, and her bowels had always acted normally. There had been no loss of weight. There had been some nocturnal frequency of micturition six months prior to her coming to hospital, but she had not experienced this since that time.

On examination of the patient, an ovoid mass about the size of an orange was felt in the left hypochondrium, which was freely movable in all directions. The tumour transmitted a pulsation, but did not give the sensation of being in any way expansile. It appeared to be hard and not cystic. The only other abnormality found on physical examination was a systolic blood pressure of 230 milli-

metres of mercury and a diastolic pressure of 130 millimetres of mercury.

A flat X-ray examination of the abdomen was carried out and revealed a circular opacity with linear calcifications in its outline, slightly to the left of the mid-line. A Casoni test, barium meal examination and full hæmatological investigation revealed no abnormality. A pre-operative diagnosis of calcifying mesenteric cyst was made.

On June 30, 1947, a laparotomy was performed and the tumour was easily located, presenting through the posterior part of the peritoneum below the transverse colon and slightly to the left of the mid-line. Transmitted pulsations, but nothing suggesting an expansile pulsation, were noted. During the attempt to locate the origin of the tumour, alarming hæmorrhage was encountered, necessitating immediate blood transfusion. The nature of the lesion was now quite clear—namely, a calcifying aneurysm. The hæmorrhage was controlled with difficulty and the patient was returned to bed in a reasonably good condition. With the recovery of the blood pressure, however, a sudden massive hæmorrhage occurred shortly afterwards, and the patient succumbed in the space of a few minutes.

Post-Mortem Examination.

An autopsy was performed twenty hours after death. The body was that of a female of slight build and poor nutrition.

In the abdomen two retroperitoneal swellings were seen in the epigastric and left hypochondriac regions, one just to the left of the mid-line, the other twelve centimetres further to the side and largely covered by the spleen. On dissection, the splenic artery was found to have two aneurysms in its course. The first, just to the left of the mid-line, was saccular in type, freely movable and 7·5 centimetres in diameter. From this aneurysm the splenic artery passed to the left and was of approximately normal diameter for a distance of five centimetres before forming the second aneurysm. This latter was also approximately 7·5 centimetres in diameter and directly adjacent to the spleen. Two arterial branches passed into the splenic substance from separate points in the wall of the aneurysm. A laminated clot was present in the distal aneurysm, and this could be removed with ease. The walls of both aneurysms were thickened and the pancreas appeared to overlie and to be adherent to the walls of both. The wall of the proximal aneurysm was calcified in some areas, and in the lining of both aneurysms patchy atheroma was present. The spleen was enlarged, measuring 17·5 centimetres by 12·5 centimetres by 4·0 centimetres, and weighed 364 grammes. Its texture was firm.

The brain showed no abnormality and the cerebral vessels appeared normal. Only very slight atheroma was present in both the thoracic and abdominal aorta, and there appeared to be no loss of elasticity.

The heart was enlarged, weighing 364 grammes. There was hypertrophy of the left ventricle, but the valves and myocardium appeared normal. The coronary arteries showed slight atheromatous change, but were patent.

The kidneys were both small and pale. The left weighed 74 grammes, the right 65 grammes. Their capsule was somewhat adherent, and reflection of this exposed a subgranular surface. Numerous small retention cysts were present in the left kidney. There was a slight increase in pelvic fat and the cut surface was pale. The vessels on the cut surface were not obvious.

Examination of the liver, gall-bladder, stomach, suprarenals and genitalia revealed no abnormality.

The Wassermann and Kline tests performed on blood taken *post mortem* produced no reaction.

Histological Examination.—Examination of sections of the walls of the aneurysms showed the walls to consist in the main of almost acellular hyaline fibrous tissue, with scattered areas of calcification. The fibrous tissue appeared to have fused and to be continuous with the pancreas. Atrophic pancreatic acini were found scattered in the dense fibrous tissue, and the remainder of the pancreas appeared to be compressed and fibrotic. There was no inflammatory cell infiltration of the wall of the aneurysm. Examination of the spleen revealed some thickening of the capsule and moderate congestion of the pulp. The myocardium contained scattered areas of fibrosis, with occasional foci showing an increase of interstitial cells. A mild degree of intimal thickening was present in the coronary arteries. In the kidneys, numerous ischæmic areas with fibrosis, atrophic glomeruli and round-cell infiltration were seen. Dilated tubules were seen adjacent to the ischæmic areas. There appeared to be an increase in the medullary connective tissue. Examination of the interlobular and arcuate arteries revealed pronounced medial and intimal thickening, and the afferent arterioles appeared thickened and many showed hyalinization of their walls. Examination of the suprarenals, medulla and *cortex cerebri* revealed no abnormality.

CASE II.—Mr. H.P., aged sixty-one years, was admitted to the Brisbane General Hospital on September 9, 1940. He complained of breathlessness on exertion present for a number of years, and of gradual swelling of the ankles and feet extending up to the abdomen for the five weeks prior to his admission to hospital. He had frequency of micturition, passing urine every hour during the day and twice each night. His weight had increased by two stone within twelve months.

On examination of the patient his pulse was regular and the heart sounds were normal. His blood pressure was 139 millimetres of mercury (systolic) and 100 millimetres of mercury (diastolic). The percussion note was dull at the base of the left lung and the breath sounds were weak over the lower lobe of the left lung. Œdema was present in the feet and scrotum, also in the sacral region.

The patient was treated for congestive cardiac failure; he progressed favourably and the œdema was subsiding. On October 6, twenty-seven days after his admission to hospital, whilst using a bed-pan, he suddenly collapsed and fell out of bed. He was pale and distressed, and his pulse was weak. On the following day he complained of pain in the abdomen and of diarrhœa. His abdomen was distended and tympanitic. Rectal examination revealed no abnormality. On October 11 the patient complained of pain in the hypogastrium, and this abdominal pain, coupled with diarrhœa, persisted. The pulse was weak and the patient appeared dangerously ill. This condition continued until November 6, when the patient became suddenly very pale and shocked, dying fifteen minutes later.

Post-Mortem Examination.

The body was very pale and extremely œdematous. Both pleural cavities were full of clear fluid, and the lungs appeared waterlogged. The heart weighed 310 grammes, the myocardium was flabby and pale, and the coronary arteries were atheromatous. When the abdomen was opened, it was found to be distended with blood-stained fluid. There was a ruptured aneurysm of the splenic artery behind the tail of the pancreas. The wall of the aneurysm was atheromatous and a laminated clot was present within its cavity. The spleen weighed 210 grammes and appeared normal on macroscopic examination. The surface of the liver was nodular, and on examination of sections, advanced "nutmeg change" and fibrosis were apparent. The kidneys appeared normal, apart from being pale.

No histological examination was made.

Discussion.

The first case presented a diagnostic problem which was made more difficult by the great mobility of the mass. The complete absence of subjective symptoms was striking, in view of the size of the aneurysms. The concomitant hypertension and moderate degree of atherosclerosis suggest that the condition was probably due to arterial degeneration; but the possibility of a localized congenital defect of the vessel wall could not definitely be excluded.

In the second case, there appeared to be no symptoms at all referable to the splenic aneurysm, and if these were present they were masked by those of advanced congestive cardiac failure. Unfortunately there is no record of the size of the aneurysm. I regard this second case as an example of the "two stage" rupture of an aneurysm of the splenic artery. It would seem that the primary rupture took place whilst the patient was straining at stool and caused his initial collapse, and that the final or fatal hæmorrhage occurred four weeks later. It was only after his initial collapse that symptoms referable to the aneurysm occurred, such as abdominal distension and pain.

The ætiology of the second aneurysm was almost certainly atherosclerosis with associated hypertension.

Each of these cases is in itself of interest and serves to stress the many and varied ways in which these aneurysms of the splenic artery may present themselves, and the diagnostic problems they may provide.

Summary.

1. One hundred and forty-five cases of aneurysm of the splenic artery have previously been recorded in the litera-

ture. Under the age of forty-five years a preponderance of females appears to be affected. These aneurysms are not infrequently associated with pregnancy.

2. Of the various ætiological factors, arterial degeneration, congenital defects of the vessel wall and endocarditis are the most likely. Syphilis, trauma, splenomegaly, increased portal pressure and *periarteritis nodosa* are comparatively rare causes. The association of pregnancy with the formation of these aneurysms is more apparent than real.

3. The clinical features vary according to whether rupture has occurred or not. Even in the former case, the clinical picture is not clear cut, as the aneurysm may rupture by a single or "two stage" process.

4. Even in the absence of pregnancy, the outlook is very poor.

5. Before rupture of the aneurysm, the treatment of choice is splenectomy with removal of the segment of the splenic artery from which the aneurysm has arisen. After rupture, heroic surgery and blood transfusion are needed.

6. A case of twin aneurysms occurring in the splenic artery of a single woman is reported. The patient presented a diagnostic problem, and fatal hæmorrhage from rupture of one of these aneurysms occurred during exploratory laparotomy. A record is given of the post-mortem and histological findings.

7. The second case report is of the rupture of an aneurysm of the splenic artery in an elderly man, which occurred whilst he was undergoing treatment for congestive cardiac failure. It is considered that this rupture was of the "two stage" variety. Details of the post-mortem findings are given.

Acknowledgements.

I wish to thank Dr. John Lynch, of Brisbane, for clinical details of the first case and for his assistance in the compilation of this paper. My thanks are also due to Dr. A. Pye, Superintendent of the Brisbane General Hospital, for access to the hospital records, and to Dr. E. H. Derrick for his helpful criticism and advice. The photograph was taken by Mr. E. Bagnall, of the University of Queensland Medical School.

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THE URINARY EXCRETION OF 17-KETOSTEROIDS AND OF CORTICOSTEROID-LIKE HORMONES BY THE NEWBORN INFANT.

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ALTHOUGH the relatively enormous fœtal adrenal gland and its rapid neonatal involution have attracted the interest of many workers, the exact nature of its physiological function has not yet been demonstrated. An androgenic role has been assigned to it by Grollman(1) and Broster(2) on the basis of the similarity of the staining reaction of the fœtal adrenal tissue to that found in adult hyperplasia associated with virilism; but Carnes(3) was not able to extract an androgenic substance from the fœtal tissue, and Talbot(4) in an examination of the 17-ketosteroid excretion in the urine of normal children found in two infants less than one year of age an output of less than one milligramme in twenty-four hours. The hypothesis that the fœtal adrenal gland protects the unborn child in some way against the high titres of maternal hormones, which might tend to modify the genetic factor in sex differentiation, would be difficult of experimental proof in the human subject. Carnes(3) reports high œstrogen levels in the fœtal adrenal, but does not venture an opinion as to whether they are fœtal or maternal in origin. Bruch and McCune(4) have examined the blood of infants from birth to the age of twenty-five days, determining hæmatocrit values, specific gravity, and serum protein, sodium, total base and non-protein nitrogen levels, and find that they are within normal limits and remain constant at a time when the fœtal adrenal is undergoing rapid involution, so that marked anatomical change in the fœtal adrenal is not accompanied by alterations in the serum electrolyte pattern such as those associated with destructive lesions of the adult glands. Smith,(5) in a discussion on the fœtal adrenal, cites the experiments of Stewart and Rogoff(6) on the adrenalectomized dog as proof that the fœtal cortex produces an active hormone which can pass the placental barrier to protect the mother from the effects of adrenalectomy. An examination of the experiment shows that a pregnant bitch survived bilateral adrenalectomy for sixteen days without treatment and died of cortical failure after her pups were born. However, one other bitch, not pregnant, also survived bilateral adrenalectomy for fifteen days without treatment, so the pregnancy may not be the only factor concerned.

The urinary excretion of the 17-ketosteroids and the corticosteroid-like hormones has been used in the evaluation of the degree of activity of the adult adrenal. It was thought that a similar series of figures for the newborn infant might throw some light on the function of the fœtal adrenal and also supply a normal standard which might be useful for comparative purposes in the investigation of infants showing developmental sexual anomalies. Accordingly, 17-ketosteroid and corticosteroid-like hormone excretion in the twenty-four hour specimen of urine has been determined in a series of 19 normal babies born in King George V Memorial Hospital for Mothers and Babies.

The chief difficulty in carrying out quantitative metabolic studies on the newborn is the collection of a full twenty-four hour specimen of urine. For the purpose of this experiment only normal boys were used, as a technique of collecting satisfactory specimens from girls has not yet been perfected.

The ages of the infants range from a few hours to eight weeks. Two of the older babies were premature and were aged six and eight weeks respectively when the urine specimens were examined. To collect the urine a form of external catheterization was used as follows. The extreme end of a tapered centrifuge tube, nine-sixteenths of an inch in internal diameter, was cut off and the narrow portion was pushed into a piece of rubber or plastic tubing about two feet long. The wide end of the centrifuge tube was fitted over the penis and attached to the anterior

TABLE I.

Twenty-four Hour Excretion of 17-Ketosteroids in the Urine of New-born Infants.

Case.	Infant's Age.	Weight.	Urine Volume. (Millilitres.)	Total 17-Keto-steroids. (Milligrammes.)	α Group. (Milligrammes.)	β Group. (Milligrammes.)	Clinical Notes.
		lb. oz.					
I	24 hours.	8 0	76	0·62	0·37	0·25	
II	48 hours.	7 8	70	0·25	0·25	Nil	
III	30 hours.	5 11	32	0·13	0·13	Nil	
IV	96 hours.	6 0½	133	0·67	0·50	0·17	
V	96 hours.	4 13½	206	0·17	0·16	0·01	
VI	84 hours.	—	73	0·37	0·37	Nil	
VII	28 days.	8 11	235	0·25	0·13	0·12	Has hare-lip.
VIII	24 hours.	7 11	73	2·50	2·38	0·12	Cæsarean section; no labour.
IX	48 hours.	9 13½	56	3·37	3·25	0·12	Cæsarean section; trial labour.
X	96 hours.	8 8	116	0·75	0·48	0·27	Cæsarean section; no labour.
XI	30 hours.	12 0	195	1·70	—	—	Cæsarean section; mother diabetic.
XII	30 hours.	—	40	0·25	—	—	Cæsarean section.
	5 days.	—	180	1·25	—	—	Same baby, four days later.

abdominal wall with adhesive strapping. The free end of the rubber tubing was then tied into a specimen bottle hung on the side of the cot. The tubing can be clamped if it is necessary to take the baby from the cot.

Analysis of the specimens was begun as soon as the collection of urine was completed, so no preservatives were added. The 17-ketosteroids were determined by the method of Cahen and Salter,[7] the α and β groups being separated by digitonin precipitation according to the technique of Bauman and Metzger.[8] The corticosteroids were estimated by the method of Talbot, Saltzman, Wixom and Wolfe.[11] When the total volume of urine was less than 250 millilitres the whole specimen was taken for analysis.

The results are given in Tables I and II, together with the ages and weights of the babies. As the number of cases examined is small, the figures have been given in full and no mean figure or variation has been determined.

It will be seen that in Table I the excretion of 17-ketosteroids is less than one milligramme in twenty-four hours in all except four instances. This agrees with the findings of Talbot,[9] who in a series of 17-ketosteroid levels in the urine of normal children gives figures of less than one milligramme in twenty-four hours in two children below the age of one year, and at three years of age found a twenty-four hourly excretion of 0·15 milligramme. The four babies with high levels showed no abnormality of sexual development. Each of these babies was delivered by Cæsarean section; but two other babies delivered in this way had low androgen levels. It was thought that part of the 17-ketosteroid might be maternal in origin; one steroid, cholesterol, has been shown by Goldwater and Stetten[10] to pass the placental barrier, so it is reasonable to suppose that other substances of similar chemical structure do so. Cæsarean section allows less time for adjustment between the ante-natal and post-natal metabolism, between parasitism and the free living state, than does normal labour, in which uterine contractions interfere with the maternal-fœtal circulation relationship before birth is accomplished. However, one baby (Case XII), who was examined thirty hours after delivery by Cæsarean section, had a low 17-ketosteroid excretion at that time; five days later the figure was five times as great. Moreover, the highest level of 3·37 milligrammes in twenty-four hours was obtained forty-eight hours *post partum* from a baby whose mother had had a trial labour and was delivered by Cæsarean section for disproportion. The figures found are not in favour of an increased androgenic function for the adrenal of the fœtus or newborn and are not high enough to suggest that the fœtal contribution of androgens to the mother, at least at the end of pregnancy, would be sufficient to account for the different levels of androgenic excretion between mothers of male and mothers of female babies found by Burrows, MacLeod and Warren.

TABLE II.

Twenty-four Hour Excretion of Corticosteroid-like Substances in the Urine of New-born Infants.

Case.	Infant's Age.	Urine Volume. (Millilitres.)	Corticosteroid-like Substances. (Milligrammes.)	Clinical Notes.
A	6 weeks.	308	0·06	Premature infant, making normal progress.
B	6 days.	198	Nil	Meckel's diverticulum and hernia.
C	18 days.	215	0·05	Had *icterus gravis*.
D	3 days.	51	0·2	
E	23 days.	350	0·05	
F	8 days.	124	0·05	
G	8 weeks.	110	0·1	

Only small amounts of dehydroisoandrosterone were found even in those cases in which the total amounts of 17-ketosteroids were relatively high.

The corticosteroid-like hormone levels are low in every instance. In one case no reduction of the reagent occurred. The mean figure for the seven determinations is 0·07 milligramme. Talbot[11] gives an average daily excretion of 0·24 milligramme for adults, approximately 0·003 milligramme per kilogram of body weight. The figures in Table II for an average seven-pound baby work out at 0·02 milligramme per kilogram of body weight. However, if the difference in the relative sizes of the adult and fœtal adrenals is used for comparison, the adult excretes 0·024 milligramme per gramme of adrenal tissue in twenty-four hours and the newborn baby excretes 0·015 milligramme per gramme of adrenal tissue. If these corticosteroid-like substances in the urine are an index of the cortical activity of the adrenal, then the newborn baby has an adequate but not an increased adrenal function. This finding is in agreement with the results of the investigation of the electrolyte pattern by Bruch and McCune, quoted above.

Two other points were noted in the course of the examination. In no instance did the red indigoid pigment so commonly found in adult urine appear in the infants' urine. As this pigment is said to be associated with intestinal putrefaction and has been particularly correlated with protein ingestion by Rimington,[12] its absence from the urine of the newborn baby is not surprising.

One baby had lipuria for a short time (Case IX). This baby was forty-eight hours old when the urine was collected. He weighed nine pounds fourteen and three-quarter ounces and had been delivered by Cæsarean section. He passed 56 millilitres of urine in twenty-four hours, of a white, milky appearance. It was not contaminated by fæces, and the turbidity was not due to phosphaturia. Microscopic examination showed many fat droplets and a few round, clear cells. The baby passed no other specimen of chylous urine and made perfectly normal post-natal progress. Albuminuria is a physiological finding in the newborn and a glomerulus which is permeable to the albumin molecule may possibly allow the passage of microns of fat.

Summary and Conclusions.

The 17-ketosteroid excretion in the urine has been determined in a series of 12 normal male newborn infants. The levels do not suggest an excessive androgenic function of the adrenal cortex. Four babies delivered by Cæsarean section excreted comparatively large amounts of androgens.

The amount of corticosteroid-like hormone excreted in the urine of seven normal male newborn infants has been found to approximate in value per gramme of adrenal tissue to figures given for normal adults.

One instance of transient chyluria has been noted in an apparently normal baby.

Acknowledgements.

I should like to thank the honorary medical officers of the King George V Hospital for their permission to examine babies under their care; Dr. L. Cains for assistance in collecting material for the earlier part of the investigation; and the sisters and nurses of the nurseries for their cooperation and for the meticulous care they took in the collection of the urine specimens.

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THIAMINE IN PINK DISEASE.

By EDGAR H. M. STEPHEN,

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I AM impelled to give a brief account of the satisfactory results following the use of thiamine in pink disease, as eight consecutive cases have appeared to me to demonstrate its value.

I have always made a practice of administering what seemed to me an adequate dose of vitamin *B* orally to all patients with pink disease, either as "Ryzamin B" or as "Vibex", but in these cases the dose has been ten milligrammes by intramuscular injection every four days. Dr. R. T. Dalton, of Singleton, directed a young friend of his to me, on her return to Sydney after a holiday. The child was definitely suffering from pink disease, and her anorexia, irritability and loss of weight were causing deep concern. Dr. Dalton treated her with intramuscular injections of vitamin B_1. Within a few days there was a marked amelioration in the symptoms, and this improvement was maintained.

I was much impressed with the results obtained in so short a time and determined to follow a similar procedure. I think the doses I have used have been smaller than in the case described, but they have proved successful. The preparation I have advised is Glandular Products "Thiavite" (Glandular Preparations, Proprietary, Limited), which contains 50 milligrammes of thiamine per millilitre. Three minims have been injected intramuscularly every fourth day. The medical men in charge of these eight patients have reported most satisfactory progress and were naturally gratified. In one instance I found it advisable to give an injection daily for three doses. As this child was admitted to hospital, this was easy to carry out, and he now eats and sleeps and is gaining condition. The duration of illness prior to the institution of the treatment in these cases varied from two to twelve weeks, and I do not consider that there was any doubt as to the diagnosis. All but one of the patients were treated in their own homes.

Dr. Dalton has now let me have the dosage he used. The doses were 25 milligrammes for the first four doses, then ten milligrammes by intramuscular injection every second day.

Addendum.

Since the foregoing report was written, I have been shown a comment on page 896 of the *British Medical Journal* by Dr. G. M. B. Hales, of Sydney. Dr. Hales reports having treated patients suffering from pink disease with intramuscular injections of liver extract and vitamin B_1, fifteen milligrammes twice a week. Loss of irritability after the third or fourth injection was noted, especially if the patient was treated early. It was rare for more than twelve injections to be required, and recovery was generally complete in about eight weeks. Dr. Hales tells me that in his experience better results have been obtained if 1·0 millilitre of "Anahæmin" was combined with the use of vitamin B_1.

Reports of Cases.

MESENTERIC CHYLANGIOMA IN A NINE MONTHS OLD BABY.

By THOMAS F. ROSE,

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CHYLANGIOMA OR CHYLOUS CYST is one variety of mesenteric cyst. That it is sufficiently rare to merit interest is shown by the fact that Beahrs and Judd[3] found only seven instances of chylous cysts in 174 cases of cysts of the omental, mesenteric and retroperitoneal regions in over 1,000,000 admissions to the Mayo Clinic. Only two cases of chylous cysts have been previously reported in the Australian literature since 1927.[9,10]

Clinical Record.

A female child, aged nine months, was admitted to the Royal North Shore Hospital of Sydney with the provisional diagnosis of an appendiceal abscess. Her mother stated that the child had been constipated since birth, and that at the age of five months she had had a day's illness in which she had repeated attacks of abdominal colic, vomiting and complete constipation. This was relieved after a large bowel action. The fæces were always normal in appearance and had never contained blood or mucus. The child had then progressed normally until the twenty-four hours prior to her admission to hospital, when she again suffered from abdominal colic and complete constipation. She vomited only once just prior to her admission to hospital, and it was observed that the abdomen was distended.

Examination of the child on her admission showed her to be pale and to look ill, though she was a well-nourished

infant. The temperature was 100° F., the pulse rate was 110 per minute and the respirations numbered 30 per minute. The abdomen was distended generally, especially on the right side. Palpation, which caused obvious discomfort, revealed a rounded mass filling the right iliac fossa. This mass did not move with respiration, nor could it be moved by the palpating hand. It was dull to percussion. Auscultation of the abdomen revealed a general absence of bowel sounds. The mass was not palpable rectally and there was no blood or mucus in the empty rectum.

No abnormality was detected in the other systems, and the diagnosis of an acute appendiceal abscess was made.

Operation was forthwith performed through a transverse incision over the summit of the mass. When the peritoneum was incised, some thin, clear, yellow fluid escaped and a normal-looking appendix and caecum were seen. The incision was prolonged medially with transverse division of the right *rectus abdominis* muscle, and the mass was found to be a large multiloculated cystic tumour in the mesentery of the ileum. It was acutely inflamed, being red in colour and covered with recent fibrinous plaques which made it adherent to the anterior and posterior abdominal walls. These adhesions were easily divided by finger dissection, and the loop of ileum with the mass in its mesentery was delivered through the incision onto the

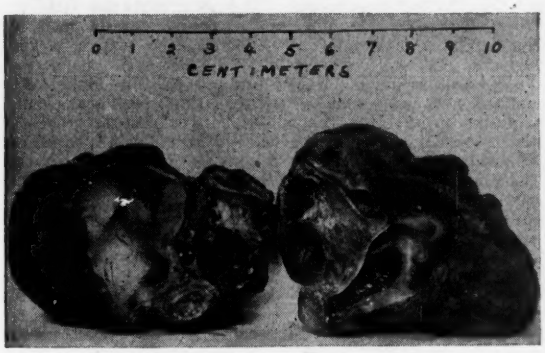

FIGURE I.
Photograph of the tumour bisected to show its multilocular structure.

abdominal wall. The tumour (Figure I) was so large in comparison to the mesentery and bowel that the ileum was stretched across it like a tape and the ileal vessels were like threads. The bowel above this loop was a little dilated, and below it was collapsed.

It was decided to remove the cyst, and in order to avoid interference with the blood supply of the bowel as many loculi as possible were aspirated by suction aspiration, a considerable quantity of material like clotted milk being withdrawn. (Through an oversight this material was not submitted to cultural examination or analysed.)

The tumour was then fairly easily removed from the mesentery, but owing to the inflammatory adhesions many of the thread-like vessels were damaged and it was seen that a section of about three inches of ileum approximately six inches from the ileo-caecal junction had lost its viability. This segment was resected and an end-to-end anastomosis was performed according to the method described by Bailey.[1] The abdominal wall was then sutured in layers without drainage.

After operation dextrose and saline solution were administered intravenously and penicillin was given intramuscularly. The child made a steady improvement, and oral feedings were given three days after operation. She was discharged home fifteen days after operation.

When examined six months later the baby was well, and her mother stated that she did not now suffer from the constipation that she had always had before the operation.

Examination of the Specimen.

On macroscopic examination (Figure I) the cyst was multiloculated and measured seven centimetres in diameter. The loculi varied greatly in size and were full of a clotted milk-like material. Many of these loculi communicated with each other, but others were completely isolated. Their walls varied in thickness and all were lined by a smooth endothelium. The tumour did not involve the bowel wall, which was easily separated from it.

Dr. C. S. Graham, morbid anatomist to the Kolling Institute of Medical Research, examined the specimen microscopically (Figure II) and reported as follows:

> This is a multiloculated chylous cyst. In addition to the large loculi obvious to the naked eye, there are many large and small "lymph" spaces lined by endothelium to be seen in the fibrous capsule of the tumour. There is an acute inflammatory reaction present indicating that the cyst has recently become infected.

It may be noted that no smooth muscle was seen in the walls of the cystic spaces such as was found in Thompson and Chambers's case,[3] nor were any giant-cell phagocytes present.

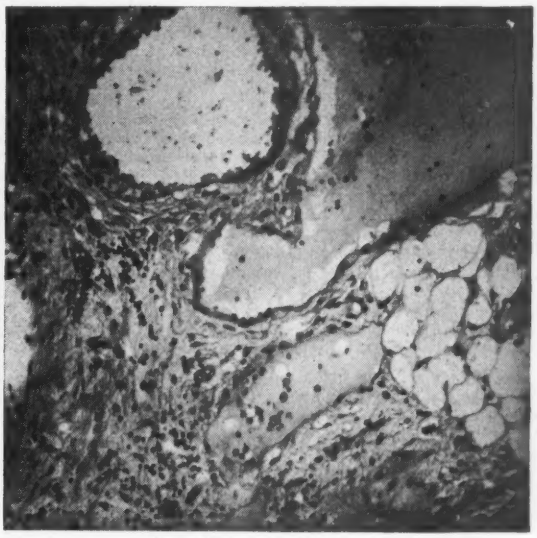

FIGURE II.
Photograph of the microscopic structure of the tumour.

Discussion.

Age Incidence.

Though mesenteric cysts as a whole are usually diagnosed in early adult life,[2] it seems that chylous cysts are more often found in children, to judge from the case reports.[4][7][8]

Ætiology and Pathology.

Lubitz and Flynn[2] classify mesenteric lymphangiomata into three groups, as follows: (i) *lymphangioma simplex*, which consists of a simple network of dilated lymph vessels and spaces; (ii) *lymphangioma cystoides*, which consists of multiple isolated cysts which have lost connexion with the lymphatics; (iii) *lymphangioma cavernosum*, which consists of numerous connecting cavities separated by thin septa. These may involve the lacteals and grow into the villi of the bowel. The case here reported fits in with this last-mentioned group of tumours, which Lubitz and Flynn[2] state are mostly found in adults. They are said to be very rare, only seven cases having been previously reported. Lubitz and Flynn[2] further state that these tumours are neoplastic and may arise in one of two ways—firstly, by homoplastic neoplasia, in which the lymphendothelium proliferates, forming

spaces communicating with preexisting lymph vessels, and secondly, and more commonly, by heteroplastic neoplasia, in which the tumour develops from a connective tissue matrix.

Diagnosis.

These tumours may reveal themselves in many ways. A freely mobile, symptomless abdominal swelling may be found on routine examination. Sometimes the patient's attention is drawn to the swelling because it may cause chronic pain.[4] Again, they may reveal themselves by the complications they cause. Obstruction is the commonest complication, and this case well illustrates how this can occur with the bowel stretched tightly across the mass swollen by inflammation. Infection is a less well recognized complication,[4] and in this instance caused the cyst to become adherent to its surroundings, so losing the classical sign of mobility and confusing the diagnosis with that of an appendiceal abscess. A fall or a blow on the abdomen may rupture such a cyst or cause hæmorrhage into its lumen.

Hinkel[3] states that a mesenteric cyst may often be diagnosed pre-operatively by radiological means. It may show as an area of increased or decreased density, or calcified areas may be seen in the walls. The bowel, examined by means of an opaque "follow-through" meal or enema, may be indented or displaced.

Treatment.

Whenever possible enucleation should be performed; it is said to carry a 9% mortality rate.[6] If bowel resection is also necessary the mortality rate rises to 25% to 30%. In this instance, as the cyst was infected, its removal was considered necessary, even though a portion of the bowel had to be sacrificed. Thompson and Chambers[8] were forced to be content with aspirating the cyst in their case, because the mass surrounded the commencement of the superior mesenteric artery in the region of the duodenojejunal junction and was obviously in too dangerous a position to allow anything more radical to be done.

Summary.

A case of mesenteric chylangioma (*chylangioma cavernosum*) occurring in a child, aged nine months, associated with infection and commencing intestinal obstruction, is reported. The diagnosis was not made prior to operation, an appendiceal abscess being simulated. The tumour was removed together with four inches of ileum. Recovery was uneventful, and a follow-up examination disclosed no further trouble.

A brief discussion of these tumours is given.

Acknowledgement.

I wish to thank the General Medical Superintendent of the Royal North Shore Hospital of Sydney for permission to publish this case report.

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(5) J. M. Lubitz and R. W. Flynn: "Chylangioma Cavernosum Mesenterii", *Surgery*, Volume XVIII, 1945, page 772.

(6) C. S. Roller: "Mesenteric Cysts: A Brief Discussion and Report of Three Cases", *Surgery, Gynecology and Obstetrics*, Volume LX, 1935, page 1128.

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(9) R. B. Wade and J. Steigrad: "A Case of Mesenteric Cyst", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, 1928, page 465.

SEVERE ZOSTER ASSOCIATED WITH LEUCHÆMIA.[1]

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THE association of zoster with leuchæmia, especially with lymphocytic leuchæmia, is fairly well recognized. About fifty cases have been reported, in about half of which "generalized zoster" has been present. However, I have seen no comment in the literature on the severity of the zoster; hence it is perhaps worth reporting that I have recently seen two patients in each of whom zoster of exceptional severity was followed by manifestations of leuchæmia.

Case I.

A man, aged sixty-five years, was referred for examination in August, 1947, on account of clumsiness, numbness and paræsthesia of the right lower limb; these symptoms had developed during the previous five or six months, but appeared to be stationary at the time of the examination. He said that he had been well till about a year previously, when he had had shingles very badly round the right side of the chest—"and chicken pox at the same time". His local doctor had said that it was the severest case of shingles he had ever seen. The skin did not heal for several months. He had had a great deal of pain for about eight months and the affected area was still very sensitive.

On examination he was found to exhibit signs of a localized organic lesion within the vertebral canal involving or affecting the spinal cord at the level of the fourth thoracic segment on the right side. Around the right side of the chest was a zone of dead-white cicatrix at the level of the nipple; but the nipple and its areola stood out in the middle of the scar quite uninvolved. It was evident that the shingles had been confluent and deeply ulcerated. There were numerous spots of pink keloid in the white cicatrix. There was no scarring left by the "chicken pox", which was presumably not chicken pox but generalized zoster—if the distinction is not too fine to be made.

A blood count showed lymphocytic leuchæmia; the white corpuscles numbered 90,000 per cubic millimetre of blood, about 96% of them being lymphocytes, nearly all mature. There was no considerable anæmia.

The cerebro-spinal fluid contained 10 cells per cubic millimetre and 30 milligrammes of protein per 100 millilitres. Myelography was performed, but no lesion within the spinal canal could be demonstrated by this means.

Case II.

A man, aged fifty-eight years, came for examination in October, 1947, on account of angina of effort.

He was seen to be very pale, and on examination enlargement of lymph glands in various situations and massive enlargement of his spleen were found. Patchy infiltration, which proved to be leuchæmic, was present in the skin of the legs, and a patch of infiltration, doubtless leuchæmic, was seen in the left retina. Several hæmorrhages were present in the retinæ.

There were numerous large and deep pock marks, confluent in places, in the cutaneous distribution of the first and second lumbar segments of the spinal cord on the left side. The patient said that two years previously he had suffered an attack of shingles, which his doctor had described as most severe, and which had caused him to be confined to bed for about six weeks; the lesions did not heal for about four months. During that time a deep ulcer had formed in his groin, through which a piece of tissue "like a bit of sheep's tongue" and as large as a walnut had been extruded.

A blood count showed lymphocytic leuchæmia; the white corpuscles numbered about 800,000 per cubic millimetre of blood, about 96% of them being lymphocytes, nearly all

[1] Read at a meeting of the Royal North Shore Hospital of Sydney Clinical Society on December 3, 1947.

mature. There was anæmia, the hæmoglobin value being six grammes per 100 millilitres of blood (42% of the standard normal figure).

Reviews.

A YEAR BOOK ON NEUROLOGY, PSYCHIATRY AND NEUROSURGERY.

"THE 1947 YEAR BOOK OF NEUROLOGY, PSYCHIATRY AND NEUROSURGERY" is divided, as its title would suggest, into three parts.[1] The first part on neurology has been edited by Hans H. Reese and Mabel G. Masten. The sections, which are divided into several subsections in the case of "Disorders of the Central Nervous System", cover a wide range of subjects. Attention is arrested by the subsection on cerebral trauma, by reference to streptomycin in the treatment of tuberculous meningitis, by the references to pathological findings in poliomyelitis, and by mention of the treatment of migraine.

The section on psychiatry has been edited by Nolan D. C. Lewis. The sections here deal with general topics, child psychiatry, schizophrenic reactions, affective reactions, miscellaneous reactions, organic reactions, toxic reactions, psychoneuroses and psychosomatic disorders, military psychiatry and therapy. Of particular interest are the references to psychosomatic factors in allergic and dermatological conditions. The editor thinks that what is known as psychosomatic medicine seems to hold great promise for the future. He admits that it is not a new specialty, but insists that its concepts are really ancient and have not been accepted—it serves "as a bridge in the persistent struggle against the old futile concept of dichotomy of mind and body".

The part of neurosurgery has been edited by Percival Bailey. The sections here cover trauma, technique, the sympathetic system, tumours, pain, peripheral nerves, intervertebral disks, hydrocephalus, epilepsy, infection and lobotomy. The last-mentioned is also referred to in the part on psychiatry.

The third part of this book will hold most interest for the specialist, but physicians and general practitioners as well as neurologists and psychiatrists will read the first with profit.

DISEASES OF METABOLISM.

"DISEASES OF METABOLISM", by G. G. Duncan, is a good book.[2] A serious attempt has been made to correlate our expanding scientific knowledge with the practice of medicine, and no one could read this book without having his outlook on metabolic diseases affected. Compared with the standard textbooks of yesterday, it is obvious that a great deal of dead wood has been cut away and the field sown with fresh seed whose germination must influence future generations of students. Our understanding of physiology is itself in the throes of a metamorphosis, and though it is impossible to expound new conceptions in a pure form—to do so would negative physiology as it would ignore "conditioned reflexes"—the authors have succeeded in making very successful grafts.

The twenty-one authors who make contributions, each well known in his own field, have applied themselves to their task, and the result is on the whole a well-balanced edition. Some of the chapters are particularly good, and possibly the laurel wreath goes to the chapter on renal disease. From a dynamic point of view this account fits in with what one observes in patients, and it is refreshing to find that not everybody is obsessed with a fetish of trying to fit a long chronic progressive disease into the end product of the post-mortem room. There is but one criticism, and that is the statement that when the urea clearance falls to a value of 35% of normal, retention of urea begins. The calculation of urea clearance is itself a function of the level of the blood urea which is used as a denominator of the equation $C_u = \frac{U\sqrt{V}}{B}$. It is obvious that the so-called urea clearance must fall when the blood urea content rises, especially as the nominator is so influenced by the square root of the volume that the product $U\sqrt{V}$ tends to be constant. It is high time that the serious limitations of urea clearance as a clinical test of renal function should be exposed. Other chapters which appeal to us are those on carbohydrate metabolism, water balance, obesity and gout. One hesitates to extend full marks to some other chapters. Perhaps it is not fitting to criticize the shortcomings of the chapter on "Metabolic Aspects of Blood Disorders" which is confined to a little over twenty pages, but nevertheless it is disappointing to find that the author still teaches that the globin portion of the hæmoglobin molecule is available for resynthesis into hæmoglobin.

One can appreciate the difficulty of apportioning space in a book of this type so as to keep a reasonable balance and to please everybody. However, we think that in future editions the pruning knife should not be spared on the space devoted to diabetes. The practical handling of diabetes is fairly well comprehended by the majority of practitioners and long, detailed case histories seem strangely out of place in textbooks in the year of our Lord, 1948. Indeed, what is a case history? It is the resultant of what the physician extracts from a patient and the results of his treatment of the patient. Another physician with a different approach and a different personality would get another case history from the same patient. The need for and value of insulin in the treatment of diabetes arising in adult life are stressed, whereas it is common knowledge that large numbers of adult diabetics can be satisfactorily controlled indefinitely on a quantitative diet alone.

One would like to see more than thirteen pages devoted to "General Metabolism, Basal Metabolism and Conditions which Alter the Basal Metabolic Rate". This chapter could not be understood by anyone who did not have a good preliminary knowledge of the subject, and as it is the introductory chapter probably deters many from buying the book.

The second edition is well illustrated and there are numerous diagrams and schematic drawings which are very useful. The author intends the book as a text for the practitioner, but one must admit that the present level of appreciation of laboratory medicine in this country is such that very few practitioners would read the book with understanding. This book is not for the many, but the consultant physician and the teacher of modern medicine will find it an excellent text and invaluable in that it contains within its compass practically all that is worth while in the most recent advances in metabolic work.

A YEAR BOOK ON PHYSICAL MEDICINE.

IN 1938 a year book on physical therapy was added to the books of the "Practical Medicine Series" which was started in 1900. "The 1947 Year Book of Physical Medicine" is the direct descendant of the 1938 volume on physical therapy;[1] it will be welcomed by all who practise this branch of medicine and by others who may wish to know whether physical medicine has any light to shed on their particular problems. Physical medicine is not magic that can be invoked to achieve a miracle when a practitioner is at his wit's end to know what to do for his patient; neither is it a cure-all, as some appear to believe. It is a special branch of medicine which finds its application in the treatment of certain pathological conditions and in the prevention of others. Many of its procedures are based on scientific principles and it may be used unwisely in the same way that other therapeutic measures may be used by the careless or uninstructed. The volume this year is edited by Richard Kovács, who in an introduction insists that physical medicine cannot be practised apart from general medicine and surgery—"it must always be based on a definite diagnosis, and the relative value of physical measures to other medical measures must always be taken into consideration". Complicated apparatus does not form the most essential part of physical therapy. These statements of Kovács should be taken to heart by both physical therapists and practitioners of other branches of medicine.

[1] "The 1947 Year Book of Neurology, Psychiatry and Neurosurgery"; Neurology—edited by Hans H. Reese, M.D., and Mabel G. Masten, M.D.; Psychiatry—edited by Nolan D. C. Lewis, M.D.; Neurosurgery—edited by Percival Bailey, M.D.; 1948. Chicago: The Year Book Publishers Incorporated. 7" x 4¾", pp. 702, with many illustrations. Price: $3.75.

[2] "Diseases of Metabolism, Detailed Methods of Diagnosis and Treatment: A Text for the Practitioner", edited by Garfield G. Duncan, M.D., with contributions by various authors; Second Edition; 1947. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9¾" x 6¾", pp. 1064, with many illustrations, some of them coloured. Price: 84s.

[1] "The 1947 Year Book of Physical Medicine", edited by Richard Kovács, M.D.; 1948. Chicago: The Year Book Publishers Incorporated. 7" x 4¾", pp. 432, with illustrations. Price: $3.75.

The volume is divided into two main parts; the first deals with physical therapeutic methods and the second with applied physical therapy. At the outset readers are reminded that physical medicine must remain highly empirical until advances have been made in the understanding of the interaction of temperature, light, electricity, magnetism and penetrating radiations with tissue and cellular systems. A plea is also made for increased attention in medical curricula to the science of biophysics. In the first part also electric injury is discussed as a unique biological phenomenon. Electricity produces in the body changes which are basically different from electric burns or other thermodynamic changes, both in appearance and in biological characteristics. Evidence is produced to show that the passage of an electric current has a mechanical rather than a heat effect. It is of interest to note, and should be widely known, that in a number of cases of apparent death from electrical injury almost immediate recovery of the central nervous system has followed lumbar puncture. There is an interesting reference to the use of ultrasonotherapy in certain conditions. This has been done by André Denier. This author has an ultrasonoscope by which it is held that an abnormal organ or a pathological structure can be delimited on the surface with precision and a growth can be traced to its organ of origin through its pedicle. Ultrasonotherapy has been used effectively in such conditions as Raynaud's disease, Bürger's disease, asthma and certain spasmotic conditions. The editor points out that Denier is the only clinician to report on work of this kind, that much of it is clinically uncontrolled, but that it offers interesting possibilities. Many other items of interest could be selected, but there is no need for this. It must suffice to state that the subject appears to be well covered and that readers will profit from perusal of the book. They will need to be critical. The editor does not add as many footnotes as editors of some of the other year books do, but he can be direct and even devastating. For example, he designates one author's description as uncritical and adds that its implication that the therapy described brings about a remission "is rather speculative". We predict that this book will be widely read and appreciated by those for whom it is intended.

A YEAR BOOK OF OBSTETRICS AND GYNÆCOLOGY.

AMONG the other books of the "Practical Medicine Series" there has appeared "The 1947 Year Book of Obstetrics and Gynecology", edited by J. P. Greenhill.[1] As usual it consists of a series of abstracts from medical journals published during the year together with occasional comments by the editor. As a matter of fact the editor has in this volume been particularly generous with his comments. This is often valuable and gives the reader a more reliable account of a subject than he would otherwise obtain. For instance, on the subject of nutrition in pregnancy—the effects of dietary deficiency in pregnancy and detection and treatment of nutritional deficiency diseases—reference is made to an article by C. F. Vilter, D. Morgan and T. D. Spies. The edition follows the abstract of this paper (it is a fairly long abstract) by nearly two pages of comment in which he shows from the literature that knowledge on the subject is incomplete and that caution must be exercised in coming to conclusions on certain aspects of the subject. Again in the gynæcological section, following an abstract of a paper by H. Guggisberg on the treatment of uterine myomata, in which conservation of uterine tissue in relation to the maintenance of ovarian function is advocated, the editor enlarges on the discussion, presenting the views of several authors as well as his own.

The book is divided into two main parts dealing respectively with obstetrics and gynæcology. In the first part there are sections on pregnancy, labour, the puerperium and the newborn. In the last-mentioned there are several references to the Rh factor, to Rh iso-immunization and so on. The sections in the part of the work devoted to gynæcology deal with general principles, diagnosis, infertility, ectopic pregnancy, operative technique, infections, benign tumours, special ovarian tumours, malignant tumours, menstruation and its disorders and endocrinology. In the first of these are some important references to pelvic pain and in the last endocrine therapy is discussed in several abstracts.

This is a volume for both the specialist and the general practitioner; it is the kind of book they both need.

[1] "The 1947 Year Book of Obstetrics and Gynecology", edited by J. P. Greenhill, B.S., M.D., F.A.C.S.; 1948. Chicago: The Year Book Publishers Incorporated. 7" × 4¾", pp. 590, with many illustrations. Price: $3.75.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Private Enterprise or Government in Medicine", by Louis Hopewell Bauer, A.B., M.D., F.A.C.P.; 1948. Oxford: Blackwell Scientific Publications, Limited. Springfield: Charles C. Thomas. 9" × 7", pp. 212, with two illustrations. Price: 25s. net.

"An Introduction to the Principles and Practice of Homoeopathy", by Charles Wheeler, M.D., B.S., B.Sc. (London), and J. D. Kenyon, M.B., Ch.B., L.R.C.P., B.Sc. (Victoria); Third Edition; 1948. London: William Heinemann (Medical Books), Limited. 8½" × 6½", pp. 380. Price: 21s. net.

"An Atlas of Anatomy", by J. C. Boileau Grant, M.C., M.B., Ch.B., F.R.C.S. (Edinburgh); Second Edition; 1947. London: Baillière, Tindall and Cox. 11" × 8½", pp. 522, with 591 illustrations. Price: 55s. net.

"Oral Vaccines and Immunization by Other Unusual Routes", by D. Thomson, O.B.E., M.B., Ch.B., D.P.H., R. Thomson, M.B., Ch.B., assisted by J. T. Morrison, M.D., D.P.H. (Aberdeen); 1948. Edinburgh: E. and S. Livingstone, Limited (for the Pickett-Thomson Research Laboratory). 11" × 9½", pp. 344. Price: 42s. net.

"The Acute Bacterial Diseases: Their Diagnosis and Treatment", by Harry F. Dowling, M.D., F.A.C.P., with the collaboration of Lewis K. Sweet, M.D., and Harold L. Hirsh, M.D.; 1948. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9" × 6", pp. 474, with illustrations, some of them coloured. Price: 45s. 6d.

"Clinical Diagnosis by Laboratory Methods: A Working Manual of Clinical Pathology", by James Campbell Todd, Ph.B., M.D., and Arthur Hawley Sanford, A.M., M.D., with the collaboration of George Giles Stilwell, A.B., M.D.; Eleventh Edition; 1948. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9½" × 6½", pp. 966, with many illustrations, some of them coloured. Price: 52s. 6d.

"The Medical Clinics of North America" (issued every two months); 1948. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. Nationwide Number; 9" × 6", pages 258, with illustrations. Price: £5 5s. (paper binding) and £6 6s. (cloth binding) per clinic year.

"The 1947 Year Book of Pathology and Clinical Pathology"; Pathology—edited by Howard T. Karsner, M.D., assistant editor, Herbert Z. Lund, M.D.; Clinical Pathology—edited by Arthur Hawley Sanford, M.D.; 1948. Chicago: The Year Book Publishers Incorporated. 7" × 4¾", pp. 558, with illustrations. Price: $3.75.

"Medical Education", by Ffrangcon Roberts, M.D.; 1948. London: H. K. Lewis and Company, Limited. 8½" × 5½", pp. 188. Price: 12s. 6d.

"Cardiovascular Diseases: Their Diagnosis and Treatment", by David Scherf, M.D., F.A.C.P., and Linn J. Boyd, M.D., F.A.C.P.; Second Edition; 1948. London: William Heinemann (Medical Books), Limited. 8½" × 5½", pp. 756, with illustrations. Price: 63s.

"Veterinary Education: An Inaugural Lecture", by W. I. B. Beveridge; 1948. Cambridge: The University of Cambridge. 7½" × 4¾", pp. 40. Price: 1s. 6d.

"A Manual of Practical Obstetrics", by O'Donel Browne, M.B., M.A.O., M.A., Litt.D., F.R.C.P.I., F.R.C.O.G.; Second Edition; 1948. Bristol: John Wright and Sons, Limited. London: Simpkin Marshall (1941), Limited. 8¾" × 5¾", pp. 676, with illustrations. Price: 35s.

"Modern Clinical Psychiatry", by Arthur P. Noyes, M.D.; Third Edition; 1948. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. 9½" × 6", pp. 536. Price: 42s.

"The Surgical Clinics of North America" (issued every two months); 1948. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical) Proprietary, Limited. New York Number. 9" × 6", pp. 286, with illustrations. Price: £5 5s. (paper binding) and £6 6s. (cloth binding) per clinic year.

"Hutchison's Food and the Principles of Dietetics", by V. H. Mottram, M.A. (Cantab.) and George Graham, M.D. (Cantab.), F.R.C.P. (London); Tenth Edition; 1948. London: Edward Arnold and Company. 8½" × 5½", pp. 754. Price: 21s.

"Human Nutrition", by V. H. Mottram, M.A. (Cantab.); 1948. London: Edward Arnold and Company. 7½" × 4¾", pp. 160. Price: 6s. 6d.

"Thyroid Enlargement and Other Changes Related to the Mineral Content of Drinking Water: With a Note on Goitre Prophylaxis", by Margaret M. Murray, J. A. Ryle, Beatrice W. Simpson and Dagmar C. Wilson; 1948. Medical Research Council of the Privy Council, Memorandum Number 18. London: His Majesty's Stationery Office. 9½" × 6", pp. 40. Price: 9d.

The Medical Journal of Australia

SATURDAY, JULY 31, 1948.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

A TUBERCULOSIS SURVEY IN ENGLAND.

WHEN the fifth session of the Australasian Medical Congress (British Medical Association) was held at Adelaide in 1937 great prominence was given to discussions on tuberculosis; tuberculosis was in fact the central *motif* of the gathering. The discussions gave a stimulus to Australian workers on the subject, as indeed they might have been expected to do, but as practitioners of medicine know full well, officialdom is slow to move when money has to be spent on a long-range health objective, and we cannot claim that the governments or the people of the several States understand what warfare against tuberculosis really means. In a few weeks' time the sixth session of Congress is to be held at Perth and pulmonary tuberculosis is to be the subject for discussion at the plenary session. The subject will be introduced by the President, Dr. D. M. McWhae; the selected speakers are four in number. Dr. H. W. Wunderly, the Commonwealth Director of Tuberculosis, will speak on the problem of control; Dr. Linley Henzell will deal with treatment; Dr. Darcy Cowan will discuss the sociological implications and rehabilitation; and Dr. Keith Hallam will direct attention to the limitations of the role of the radiologist in the diagnosis of the condition. The subsequent discussion may be expected to cover the whole range of the subject. It seems therefore that attention may with advantage be drawn to a recent publication in Great Britain which may not be readily available before Congress meets.

The Royal College of Physicians of London has published a report of what is known as the "Prophit Tuberculosis Survey, 1935-1944".[1] The survey was made possible by a generous legacy bequeathed to the Royal College of Physicians in 1932 by the late J. M. G. Prophit for research in tuberculosis. The College saw that here was an opportunity for a large-scale investigation into the natural history and epidemiology of the disease in the young adult population. It therefore formed a special committee, of which Lord Moran, the President of the College, was chairman, and appointed a scholar to carry out the work. The first scholar was Dr. F. Ridehalgh; he was succeeded by Dr. I. M. Hall and later by Dr. M. Daniels and Dr. V. H. Springett. The report comprises the data collected by the scholars and also the general conclusions and recommendations approved by the committee. The preface by Lord Moran is comprehensive and full of information.

Lord Moran explains that the main object of the survey was to try to determine whether it is possible to pick out those persons or groups or persons most likely to develop tuberculosis. Between the beginning of 1934 and the end of 1943 observations were made on no less than 10,000 presumably healthy young adults of both sexes. Five groups were selected for study—contacts, controls, nurses, medical students and naval training establishment entrants. Contacts were drawn from persons attending tuberculosis dispensaries who were known to be exposed to a relatively heavy degree of infection because they lived in a family to which belonged a patient with tuberculosis. Controls consisted mainly of office workers who might be regarded as average citizens exposed to the ordinary risks of tuberculous infection inseparable from urban life. Nurses were employed in large general hospitals. They were classified into two groups. One group (A) came from hospitals admitting all types of patients, including those with chronic and advanced tuberculosis; the other (B) came from hospitals more selective in the type of patients. Medical students comprised students in pre-clinical years and also some from clinical years who would come into contact with tuberculosis patients in hospital. Naval training establishment entrants or "Navy boys", because of their youth and the short time for which they could be under observation, were rather a special group. Originally it was intended to observe 5000 persons in each group; the intervention of the war in 1939, however, led to curtailment of the scope of the survey. The result was that a more intensive study was given to nurses than to other groups. The numbers of persons studied in the different groups were as follows: controls, 881 females and 722 males; Navy boys, 907; medical students, 306 females and 2174 males; nurses, 3085 females in Group B and 2006 females in Group A; contacts, 576 females and 484 males. The number of nurses was not so great in comparison with other groups that the findings should be applied to nurses only. Each subject was submitted to (*a*) a skin test (Mantoux) with graded doses of tuberculin to discover whether previous infection with the tubercle bacillus had occurred or not, and (*b*) an X-ray examination to show whether there was evidence in the lungs of healed or possibly active tuberculosis. Controls, nurses and medical students had a mean age of twenty-one years on entry into the survey; contacts had a mean age rather under eighteen years and Navy boys a mean age of just under sixteen years. Controls, nurses and medical students were drawn from relatively "privileged groups".

The percentages of persons reacting to 1·0 milligramme or less of old tuberculin were as follows: contacts, 93; controls, 85; medical students, 84; nurses, 80; and Navy boys, 64. It is concluded that for young adults in England the incidence of tuberculin sensitization is about 85% for males and 82% to 83% for females. The incidence of sensitization increased with age; it was 7% to 10% higher

[1] "Tuberculosis in Young Adults: Report of the Prophit Tuberculosis Survey, 1935-1944" (Royal College of Physicians), by M. Daniels, M.D., D.P.H., F. Ridehalgh, M.A., M.B., B.Ch., M.R.C.P., V. H. Springett, M.B., B.S., and including the results of I. M. Hall, M.B., B.S., M.R.C.P.; 1948. London: H. K. Lewis and Company. 9¾" × 7¼", pp. 248.

at twenty-three years of age than at eighteen. As might be expected, contact with known tuberculous persons increased the incidence of sensitization. On the initial routine X-ray examination the incidence of active pulmonary tuberculosis among these young adults was found to be 0·5%; nearly 1% showed evidence of inactive tuberculosis and from 5% to 9% showed evidence of calcified foci (healed tuberculosis). Attention should be drawn to one observation in this part of the work—the absence of a linear relationship between the incidence of calcified foci and of pulmonary tuberculosis. This, it is pointed out, suggests that pulmonary tuberculosis is not always a recrudescence of endogenous infection.

When the response of a person to the Mantoux test is changed from negative to positive, a primary tuberculous infection is said to have occurred. The Mantoux conversion rate in the first year of entry to the survey was 26% for the female control group, 36% for male medical students, 54% for nurses in Group B and 80% for nurses in Group A. The rate was thought to be dependent on the degree of exposure. Lord Moran puts this the other way round by pointing out that important evidence is provided by this observation for the view that nurses have a greater exposure to infection than the average citizen. Comment is made on another observation—that of a rising sensitivity in persons most exposed to tuberculosis and of a falling sensitivity in those least exposed. This is thought to suggest that maintenance or rise in tuberculin sensitivity is due to repeated reinfection, and that in the absence of such repeated reinfection the natural course of sensitivity is to decline. In fact the Mantoux reversion rate (positive to negative) is held to be important enough to modify considerably the incidence of tuberculin sensitization in a given community.

Important observations were made on morbidity rates and the incidence of tuberculosis arising after entry into the survey. Among the 10,000 persons observed 210 cases of tuberculosis occurred; 174 of the 210 cases occurred during survey observation. The annual morbidity rates per thousand were found to be 5·5 for female controls, 9·9 for female medical students, 9·7 for Group B nurses, 13·7 for Group A nurses (exclusive of Irish and Welsh nurses in each instance because of their special susceptibility) and 32·7 for female contacts. For male controls the figure was 1·4, for male medical students it was 5·1 and for male contacts 29·7. Thus the morbidity rate was 11·4 for females and 6·2 for males. It is interesting to note that in spite of the higher morbidity, the tuberculosis mortality among nurses was lower than the rate for equivalent age groups in the general population. The importance of contact as a factor is evidenced by the observation that morbidity among medical students whose response to the Mantoux test on entry was negative was relatively high only during their clinical period of study. The nature of the response to the Mantoux test is considered in relation to the subsequent history of the subjects. For persons who were tuberculin-positive on entry into the survey the morbidity rate was 7·3 per thousand, and among those who on entry did not react to the Mantoux test the rate was 23·1. Even if persons with pleural effusion or retrogressive lesions were excluded the difference was still significant. Lord Moran states that the reason for the lower morbidity among those tuberculin-positive before observation is possibly the possession of some immunity as a result of previous infection, but he adds that it may equally well be due to their representing a survival group, susceptibles having been removed following their first infection years previously. The degree of exposure of reactors and non-reactors while under observation is also a factor in the differences in tuberculosis morbidity. "Indeed, it is only when the degree of exposure to infection is sufficiently great, and the number of fresh infections sufficiently high, that the morbidity in the initially tuberculin-negative reactors begins to exceed that in the initially positive reactors." Shortly after Mantoux conversion 8% of the subjects were found to give radiographic evidence of lung lesions; later it was found that nearly half of this 8% developed tuberculosis; on the other hand, among those in whom no such lesions were visible on conversion, only one-twentieth developed the disease. There can be no doubt about the wisdom of the statement that routine periodical examinations of young adults by tuberculin tests and chest radiography are most valuable weapons in the attack on the tuberculosis of young adults.

Mention has been made of differences in Irish and Welsh as compared with other nurses. It was found that morbidity was two and a half times as high in Irish and Welsh nurses. The difference applied irrespective of initial Mantoux state and was found in nurses of each hospital group. It is concluded that the higher morbidity among Irish and Welsh nurses is not due, or is due only to a slight extent, to the fact that a higher proportion was previously uninfected; it is not due to differences in the environment in which the morbidity occurred. The view is stated that, given equality of exposure and given equality of environment, there are between certain groups of people of different origin, marked differences in resistance to tuberculous infection. These do not constitute true "racial" differences of a permanent character. The greater resistance in non-primitive communities is probably due to development of group immunity by gradual exposure of the community to tuberculous infection; in other words the susceptibility of primitive communities is probably due to absence of such group immunity. We read that possibly group immunity is produced by survival of the least susceptible. "It seems possible that the inherited factor is not precisely one of immunity, but rather of native capacity to develop specific immunity as a result of an infection." Most of the lesions discovered during the observation were "minimal", but many became sufficiently serious to require treatment. Cavities occurred in 25% of the lesions and in more than 50% of the cases sanatorium treatment was required. The factors which determine the progress or retrogression of a lesion are unknown, and this is to be the subject of a new study by the Prophit Committee.

Some of the more important aspects of this remarkable report have been mentioned. These and others not referred to deserve the careful study of all clinicians who have to deal with tuberculous patients. The most prominent factor in the production of infection is contact with a person suffering from an open infection. This is not a new observation by any means, but it needs to be preached with a new fervour. No one can read this report without being seized with a desire to engage in prevention and in case finding and in the task of creating a right attitude of mind among the intelligent members of the community.

Current Comment.

PROGRESS IN THE TREATMENT OF LEUCHÆMIA.

A GREAT deal of interest has centred during the last year or two on the occurrence of acute leuchæmia in children. The death rate from this disease was discussed in the issue of April 19, 1947, in the light of a communication by M. S. Sacks and I. Seeman. These authors, whose article was published in *Blood*, analysed the statistics for the United States of America since 1900. It was found that the recorded rate had risen continuously from 1900 and that since 1930 the rise had been steadily accelerating. The rise seemed rather too great to be accounted for by improvements in diagnostic methods. In an editorial in the same journal, William Damashek asked whether leuchæmia was increasing. He thought that there was an overall increase in spite of the undoubted improvement in the methods of investigation of the blood as a routine measure. In regard to the cause of the condition, he wondered whether there might be something malign in the effects of some of the chemicals used not only in medicine but in everyday industrial, social and domestic life. The occurrence of such a fatal disease as leuchæmia is naturally very disturbing to the relatives of affected children, and when successful results are claimed to follow the use of newly discovered drugs, it is not unnatural for the distressed relatives to go to any lengths to obtain supplies of the vaunted remedy. During the last few weeks some publicity has been given to the hurried introduction to Australia from America of a special drug in the hope that it would save the life of a certain child. Unfortunately, the attempt was not successful. The news, however, that desperate efforts were made to secure supplies of the drug is likely to lead to other inquiries by doctors as well as by their patients. For this reason we wish to draw attention to an article dealing with the drug in question.

The article appears under the joint authorship of five workers in Boston—S. Farber, L. K. Diamond, R. D. Mercer, R. F. Sylvester, junior, and J. A. Wolff.[1] The title of the article is significant, for it deals with "Temporary Remissions in Acute Leukemia in Children Produced by Folic Acid Antagonist, 4-Aminopteroyl-glutamic Acid (Aminopterin)". These authors refer to two earlier articles by Farber, who drew attention (*a*) to what he interpreted as an "acceleration phenomenon" in the leuchæmic process, as seen in the marrow and viscera of children with acute leuchæmia who were treated by the injection of folic acid conjugates, pteroyltriglutamic acid (pteropterin) and pteroyldiglutamic acid (diopterin), and also (*b*) to an experience gained from studies on folic acid deficiency, which suggested that folic acid antagonists might be of value in the treatment of patients with acute leuchæmia. They show how studies on these lines led them to undertake investigations with what they describe as the most powerful folic acid antagonist encountered by them, 4-aminopteroylglutamic acid or aminopterin. Sixteen children were studied. Of the sixteen, ten showed clinical, hæmatological and pathological evidences of improvement of noteworthy importance and of three months' duration. In the present paper studies of five of the ten children are reported, and the histories of the five children are given in some detail. In one case, that of a boy aged seven years, the condition was progressing in spite of treatment. The patient became critically ill with a temperature of 106° F. A remission occurred after treatment with two folic acid antagonists. On two occasions aminopterin produced good clinical and hæmatological remissions, but on both occasions the advent of stomatitis interfered with the use of the drug. In a second case, a boy of six years of age, who was critically ill, derived benefit from three weeks of daily aminopterin therapy. A third child, aged three years and eight months, had a remission of about two months' duration after the occurrence of bacteriæmia. When aminopterin therapy was started he was in a progressive phase of leuchæmia and appeared moribund. After five days' therapy an improvement in the peripheral blood picture and the sternal marrow picture took place. A fourth child had a fulminating generalized infection with bacteriæmia, and after this a clinical and hæmatological remission took place. Subsequently, during a relapse, generalized adenopathy became evident. After aminopterin therapy, the adenopathy disappeared. A fifth boy showed slow but regular progression of leuchæmia until he appeared moribund. After three daily doses each of one milligramme of aminopterin, a rapid fall in the white blood cell count took place and clinical improvement became evident.

The authors believe that aminopterin has a pronounced effect on leuchæmic bone marrow and upon the immature cells in the peripheral blood and probably also upon leuchæmic deposit in the viscera. Under aminopterin therapy the white cell count tended to return to normal. This occurred in patients whose count was initially high and also in those cases in which leucopenia was present at the onset of the therapy. The peripheral blood changes included improvement towards the normal in the value of hæmoglobin, red cell count and platelet count. The bone marrow showed changes that varied from a decrease in number to a disappearance of the leuchæmic cells and variation from hypoplasia to almost a normal pattern. Toxic effects included stomatitis with early ulceration. The authors insist that their report deals only with temporary remissions, and add that it is impossible to state whether the substance would be of value for a longer period than that covered by their studies. They think that the toxic effects may make continued use of the drug impossible. The most important statement in the conclusions of Faber and his collaborators is that they have produced no evidence that will justify the suggestion that a cure has been found for acute leuchæmia in children. It is always necessary to remember that even when a child appears moribund from leuchæmia, spontaneous remission may occur. This statement does not imply that the beneficial results reported by Faber and his fellow workers were not due to the drug exhibited. It is also necessary to remember that a disease may be arrested and not cured. What has to be realized by the practitioner is that this work is only in the experimental stage, and it must also be pointed out again that the cases described by Fabar *et alii* were the most successful in his series. The most hopeful statement that can be made is that further research of the kind that has been reported is likely to lead eventually to the solution of the riddle. If the practitioner understands exactly what has happened, he may be able to prevent anxious parents from having their hopes raised without justification.

[1] *The New England Journal of Medicine*, June 3, 1948.

TRIGEMINAL NEURALGIA.

C. E. HORTON AND A. J. BRENNAN[1] have recently reported a case of trigeminal neuralgia of approximately sixteen years' duration which occurred at the time of maturation of the spring crops. All the usual procedures were adopted, complete relief (with numbness of the face) being obtained on the left side by means of retrogasserian neurectomy. Surgical interference on the right side brought no relief of pain, the eighth nerve apparently being interrupted rather than the fifth. Pain on the right side was extreme and neither codeine nor "Demerol" brought relief. Within thirty minutes of the taking of fifty milligrammes of "Pyribenzamine" the pain completely disappeared. Subsequent acute attacks responded quickly and consistently to either "Pyribenzamine" or "Benadryl". In pain-free periods typical attacks were provoked by the administration of histamine and again relieved by antihistamine drugs. After repeated injections of histamine, however, it was found that this drug could no longer provoke an attack of trigeminal pain. The patient was discharged from hospital with instructions to take 100 milligrammes of "Pyribenzamine" each time an attack occurred. Although the report is of only one case it is too dramatic to be overlooked.

[1] *The Journal of the American Medical Association*, March 27, 1948.

Abstracts from Medical Literature.

PATHOLOGY.

Purpuric Meningococcæmia in Relation to the Shwartzman Phenomenon.

B. BLACK-SCHAFFER, T. G. HIEBERT AND G. P. KERBY (*Archives of Pathology*, January, 1947) report three experiments carried out to investigate a possible relationship between the Shwartzman phenomenon and purpuric meningococcæmia. The first experiment served to confirm and elaborate the fact that twice-washed meningococci, both living and dead, possess potent preparatory and provocatory substances capable of producing the local Shwartzman phenomenon. The second experiment, by comparing the preparatory potency of eighteen meningococcic strains, demonstrated that most of the strains (five of eight) associated with purpuric meningococcæmia fall into a unique and very potent group. The strains obtained in cases of non-purpuric meningitis produced less of the preparatory factor. In serological group distribution the two categories of meningococci were essentially identical. Bilateral necrosis of the adrenal glands with hæmorrhage was found in two animals used in this experiment. The third experiment was designed to test the response of rabbits to meningococcæmia maintained, if necessary, over a period of twenty-four hours. General cutaneous purpura was produced in a number of animals. In addition to the cutaneous lesions, one rabbit displayed marked adrenal necrosis and hæmorrhage (Waterhouse-Friderichsen syndrome). The close relationship of the general purpura to the local Shwartzman reaction was illustrated by the simultaneous appearance of both in rabbits which previous to their meningococcæmia had been prepared in one or two sites by intradermal inoculation of meningococci. Many of the animals used in the third experiment disclosed at autopsy bilateral renal cortical necrosis. Since in rabbits this lesion is recognized as characteristic of the generalized Shwartzman reaction, it is evident that washed meningococci are capable of producing not only the local but also the general phenomenon. It is believed that the Shwartzman substance acts directly or indirectly on the interlobular arteries of the kidneys, causing marked vasoconstriction, and thus initiating the sequence of events leading to bilateral renal cortical necrosis.

Sensory Nerves of the Human Heart.

ACCORDING to Edwin F. Hirsch and James F. Orme (*Archives of Pathology*, October, 1947), myelinated (sensory) nerve fibres are distributed along the coronary arteries and their branches in the myocardium. Their terminal divisions are filaments without myelin sheaths, some of which extend directly into the walls of the arteries. These sensory fibres form the anatomical pathway by which painful sensations arising in the heart are transmitted to the central nervous system. The observation that these fibres are distributed along and in the walls of the coronary arteries suggests that in the heart the painful stimuli arise in and about the coronary arteries rather than in the muscle tissues. Sensory fibres are distributed to the walls of peripheral arteries in a pattern quite similar to that observed with the coronary arteries of the heart. Accordingly, this common pattern of distribution of the sensory nerves of both the coronary and the peripheral arteries provides a common anatomical basis for the reception of pain sensations aroused by comparable conditions in all arteries of the body. The tissue factors arousing the pain sensation associated with an injury or with thrombosis are similar for all arteries, the response depending on the sensory nerve supply.

Tumours of the Carotid Body.

ACCORDING to evidence presented by Philip M. LeCompte (*The American Journal of Pathology*, March, 1948) the carotid body is a chemoreceptor, not a gland of internal secretion, and not part of the "chromaffin system". In a series of seventeen tumours of the carotid body no true chromaffin reaction was demonstrated, and no evidence for the secretion of epinephrine was obtained in assay of the fresh tissue in two instances. Tumours of the carotid body exhibit a basic pattern of nests of "chief" cells surrounded by a more or less vascular stroma. Depending on the relative amounts of "chief" cells and stroma, they may be described as "usual", "adenoma-like", or "angioma-like". The non-committal term "carotid body tumour" is preferred to other names generally used. The great majority of these tumours are both histologically and clinically benign. In view of the high operative mortality it is doubtful whether they should be removed in those cases in which ligation of the carotid arteries is necessary.

Rheumatic Carditis and Subacute Bacterial Endocarditis.

YVONNE MACILWAINE (*The Journal of Pathology and Bacteriology*, October, 1947) presents evidence to indicate that the myocardial Aschoff nodule is a specific reaction of the tissues to an attack of rheumatic fever. This lesion appears to evolve through an orderly and characteristic process of aging. The appearances at any given time are diagnostic of the phase reached in this process. A study of 34 cases of subacute and 12 cases of acute bacterial endocarditis indicated that in a large number there was evidence of rheumatic arteritis. Of more importance was the finding, in an even higher percentage, of Aschoff nodules of an age which corresponded to the clinical duration of the endocarditis. The conclusion has therefore been reached that in many cases of endocarditis, the bacterial lesion is superimposed upon a heart which is, at the time of infection, the site of an active rheumatic carditis.

Reticulocytes and their Humoral Regulation.

ACCORDING to Erik Jacobsen (*Journal of Clinical Pathology*, November, 1947) a principle capable of accelerating the ripening of reticulocytes *in vitro* can be demonstrated in plasma and in various tissues of the body. This principle consists of at least two fractions: a thermostable one, identified as tyrosine or tyrosine derivatives, and a thermolabile one. Xanthine, leucopterin and folic acid are able to act as the thermolabile factor. The greatest concentration of the thermolabile fraction is found in the stomach and duodenum. The thermolabile and thermostable fractions linked together by the activity of the reticulo-endothelial system form the principle found in plasma. An increased amount of ripening principle may be found in the plasma in some cases of increased erythropoiesis; in decreased erythropoiesis a lower content than normal may be encountered. It seems as if the number of reticulocytes in the blood varies inversely with the amount of ripening principle, at least under normal conditions.

Primary Atypical Pneumonia.

FREDERIC PARKER, JUNIOR, LESLIE S. JOLLIFFE AND MAXWELL FINLAND (*Archives of Pathology*, December, 1947) present the clinical and pathological observations in eight cases of primary atypical ("viral") pneumonia. Clinically the condition was characterized by increasing symptoms of respiratory embarrassment, diffuse moist râles and transient areas of atelectasis, but no definite signs of consolidation of the lungs. X-ray examination revealed an extensive soft miliary type of density in the lungs. The serum contained cold agglutinins. Two of the cases were complicated by acute hæmolytic anæmia, two others by severe *erythema multiforme exsudativum* and one by rheumatic heart disease with mitral stenosis. The characteristic pathological changes noted in the lungs grossly were: the congested appearance of the cut surfaces, which were studded with small greyish or dark nodules, and the hyperæmic appearance of the mucosa of the trachea and the bronchi. Histologically, there were a mononuclear type of alveolar exudate, an interstitial infiltration predominantly of plasma cells, and swelling and proliferation of the alveolar lining cells. While the bronchioles not infrequently contained polymorphonuclear leucocytes and occasionally some bacteria, their walls were infiltrated by mononuclear cells, and the epithelium was intact. Bacterial infection played a minor role except in two cases, in which there was abscess formation. Attempts to demonstrate intracellular inclusions or to isolate a virus were unsuccessful.

Normal Red-Cell Survival.

SHEILA T. CALLENDER, E. O. POWELL AND L. J. WITTS (*The Journal of Pathology and Bacteriology*, October, 1947) have followed the survival of transfused erythrocytes by the method of differential agglutination in four normal female and two normal male subjects of group A, from whom blood was removed and replaced by blood of group O. The curve of decay of the transfused cells was linear in men and appreciably curved in women. Their average lives were respectively sixty-three and fifty-four days. The relation has been deduced between the decay of transfused cells and the law of survival of the individual erythrocyte on the assumption that two types of destructive factors are normally operative. It is concluded that most red cells live for approximately the same time, one hundred and twenty days, in both men and women. Extraneous factors cause the loss of some cells before they have reached this age limit. The loss is

greater in women than in men by the equivalent of 400 millilitres of blood per month—more than can be ascribed to menstruation. The average age of cells at death is tentatively assessed at ninety to one hundred days in women and one hundred and ten to one hundred and twenty days in men.

The Pathology of Secondary Shock.

VIRGIL H. MOON (*The American Journal of Pathology*, March, 1948) presents a study which corroborates the occurrence of secondary shock in other conditions than severe trauma, burns, and after extensive surgery. It develops in sundry conditions which may cause either capillary atony or anoxia. The resulting circulatory effects in the viscera indicate widespread capillary damage. The changes varied somewhat in degree, but the pattern was consistent. The parenchymatous effects may be ascribed in part to the injurious agent itself, in part to anoxia. The severe effects seen regularly in the renal tubular epithelium probably are related to the anuria and other evidences of renal functional deficiency which accompany shock. It appears that secondary shock, like other conditions of disease, is accompanied by distinctive morphological changes which are related to its mechanism of origin and to the associated functional disturbances.

MORPHOLOGY.

The Omentum Lienale in Man.

W. DE PAULA (*The Anatomical Record*, July, 1947) reports that the frequency of occurrence of a peritoneal fold extending over the diaphragmatic surface of the spleen (*omentum lienale*, presplenic fold *et cetera*) in man was studied in 114 cadavers (white, Negro, mulatto and yellow) of both sexes. The omentum was found well individualized in 48 cases; no significant ethnical or sexual differences were noted. The *omentum lienale* is frequent enough to be included in the description of peritoneal formations in standard anatomical text-books.

Nodes of Ranvier and Incisures in Nerve Fibres.

H. E. HISCO (*The Anatomical Record*, December, 1947) studied the disposition of the nodes of Ranvier and the clefts of Schmidt-Lantermann (incisures) in nerve fibres in the hope that the investigation might contribute to our understanding of the development of these structures and the myelin sheath. Measurements of internodes and segments (lengths of fibre between two incisures or between incisures and adjacent nodes) were made in living nerve fibres from three groups of rats, young animals, normal adults, and adults, in which the sciatic nerve had been crushed thirty to forty-five days previously. Additional regenerated fibres (seven to seventeen weeks of regeneration) were examined from stained preparations. In normal adult and young nerve fibres, the internodal length is directly proportional to calibre, increasing with increasing diameter, although the internodes of the young fibres are shorter than those of the adult in any one size class. The internodes of all regenerated fibres examined are of about the same size, 300μ, irrespective of calibre. The orientation of the funnel-shaped incisures appears to be related to their position with respect to the node; there is a tendency towards reversal of direction at the node, those incisures nearest to the node pointing away from it on either side. The developmental history of the adult distribution pattern of nodes and incisures is discussed. In brief, it is suggested that all internodes are originally of approximately the same length (300μ), becoming secondarily elongated during growth of surrounding structures by passive stretching proportional to their length, the largest fibres which have the farthest peripheral connexions being subjected to the greatest stretch. Incisures develop in direct correlation with increasing calibre, and possibly also in relation to the volume of myelin per segment, but the segments so formed are also secondarily elongated passively by varying amounts in normal development. Secondary distortion is absent in regenerated fibres, since these have, for the most part, already reached their full length by the time they attain sufficient size to have nodes and incisures in the myelin sheath.

Motor End Plates.

W. V. COLE (*The Anatomical Record*, July, 1947) reports the results of studies on the size and morphological characteristics of the motor end plates and the widths and relationship of muscle fibres under normal conditions.

Endometrial Coiled Arterioles.

I. H. KAISER (*The Anatomical Record*, October, 1947) points out that in recent years in an attempt to elucidate features of menstruation which are as yet unsatisfactorily understood, attention has been directed to the blood vessels of the endometrium, particularly the coiled arteries. Coiled arteries have actually been described only in the macaque and the human. In the present communication, the author described the characteristics of the coiled arterioles of the endometrium of *Macaca mulatta*, the common rhesus monkey, which have previously been described in three dimensions, and are now presented here as they appear in routine histological section. They are found to proceed through a regular series of alterations during the menstrual cycle.

Factors Affecting Shape of Developing Skull.

S. L. WASHBURN (*The Anatomical Record*, November, 1947), in view of the possibility generally recognized for many years that the temporal muscles may affect the form of the brain case, decided to test the matter by experiment. He removed the temporal muscle on one side from 22 newborn rats so that the relation of the temporal muscle to the form of the skull and mandible could be evaluated. In 10 of the 22 animals, the long neck muscles of the same side were severed from the nuchal crest. In the absence of muscles, the temporal lines, nuchal and mastoid crests, all failed to form, and the coronoid process of the mandible was completely resorbed. The form of the interparietal bone was altered by decreased growth in the occipito-interparietal suture, caused by removal of the nuchal muscles. When the loss of muscle caused decreased growth, sutures were simpler than normal. When growth was prolonged, sutures became more complex. The internal form of the brain case was not altered by the removal of the temporal muscle alone and was affected in only the most minor way by the removal of both temporal and nuchal muscles.

Healing of Bone Defects.

C. STEINMAN (*The Anatomical Record*, December, 1947) reviews the literature concerning the process of bone repair and the role of bone transplants in the regenerative process. In the present investigation drill hole defects in the long bones of adult rabbits were repaired by intramembranous bone formation. The grafting of similar defects with viable bone fragments of eighteen-day rabbit embryos resulted in the production of a cartilagenous callus, and the defect was then repaired in part or in whole by endochondral bone formation. The use of adult bone, alive, and boiled, similarly used as graft material, did not result in cartilage proliferation.

Early Human Embryo.

G. G. ROBERTSON *et alii* (*The Anatomical Record*, January, 1948) describe a human embryo of about seventeen days. The embryonic disk is trilaminar and the ectodermal plate measures 0·462 by 0·485 by 0·047 millimetre. Differentiated axial structures in the disk include the primitive streak, Hensen's node and a short head (notochordal) process. The beginning of development of a notochordal canal is evident in Hensen's node and the notochordal process. A short cloacal membrane is present. The amniotic cavity has a volume of 0·0086 cubic millimetre, the yolk-sac cavity a volume of 0·0209 cubic millimetre. A small allantoic diverticulum extends from the yolk-sac into the body stalk. Angiogenesis is evident in the chorionic and body-stalk mesoderm. Both angiogenesis and hæmopoiesis are evident in prominent blood islands of the yolk-sac wall.

Cross Striations in Voluntary Striated Muscle.

C. C. SPEIDEL (*The Anatomical Record*, January, 1948) studied the cross striations in voluntary muscle and concludes that the concept that cross striæ are arranged in spiral fashion is not upheld. His observations demonstrate that cross striæ within each myofibril are transversely arranged.

Spermatogenetic Wave Cycle.

RAY MOREE (*The Anatomical Record*, October, 1947) describes the spermatogenetic wave cycle in the seminiferous tubules of mice, and his results are in close agreement with those described for the majority of other mammalian species. Comparison of the periods of entire cycles indicates a general and fundamental similarity of the spermatogenetic wave cycle in all mammals in which it has been thus far described. The spermatogenetic wave cycle was among the first subjects to attract attention of early students of mammalian spermatogenesis. It is suggested that information for the comparative study of these cycles may be of value in the investigation of testicular changes, such as those resulting from hybridity.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on December 4, 1947, at the Saint George Hospital, Kogarah. The meeting took the form of a number of clinical demonstrations by the members of the honorary staff of the hospital. Parts of this report appeared in the issues of July 3 and July 24, 1948.

Gynæcological Specimens.

DR. CHARLES V. SALISBURY showed specimens from three patients which demonstrated some of the uterine conditions responsible for post-menopausal hæmorrhage.

The first specimen comprised a uterus removed from a patient, aged fifty-four years, who had complained of irregular vaginal hæmorrhage for a period of six months. The climacteric had occurred at the age of forty-nine years, and at that time she had had severe menopausal symptoms and had been treated with one of the œstrogenic hormones given orally. This treatment had been continued intermittently for a period of five years. On examination of the patient the uterus had been found to be large and globular, and an extensive deep erosion of the cervix was present. The fornices appeared clear. At curettage a large amount of tissue was removed, and as this tissue had the macroscopic appearance of neoplastic tissue, a total hysterectomy and a bilateral salpingo-oophorectomy were performed. Examination of sections of the uterine scrapings revealed that the tissue was hyperplastic endometrium, and there was no evidence of malignancy. Dr. Salisbury said that post-menopausal hæmorrhage secondary to œstrogenic therapy was frequently encountered in the out-patient department, and in his experience was the commonest form of post-menopausal hæmorrhage. Frequently hormone treatment was given in large dosage and for prolonged periods. It was important to prescribe the minimum amount of the œstrogenic hormone which would keep the patient free from symptoms, and to review and if possible reduce the amount at frequent intervals. It was also important to prevent a continuous high level of œstrogen in the blood stream, and this could readily be achieved by omitting the hormone for three or four days during each month. Such patients usually required admission to hospital for a diagnostic curettage in order to distinguish the condition from carcinoma of the body of the uterus.

The second specimen presented was a uterus of a single woman, aged forty-nine years. The menopause had occurred at the age of forty-five years, and she had been symptom-free until three weeks before her admission to hospital, when she had had a severe vaginal hæmorrhage. This hæmorrhage had increased in severity and had been continuous since then. On her admission to hospital her hæmoglobin value was 40%, and she was given two litres of blood and adequate amounts of iron for ten days before operation. The uterus was large, smooth and globular, the cervix was healthy and the os was of average size. After dilatation of the cervix a large mass which filled the uterine cavity was felt with the curette, and this was diagnosed as a pedunculated submucous fibromyoma. Subtotal hysterectomy was performed. Examination of the specimen revealed a large submucous fibromyoma four and a half inches in diameter, which completely filled the uterine cavity, and this condition was responsible for the severe uterine hæmorrhage. Dr. Salisbury said that the interesting features of this case were the sudden onset of severe hæmorrhage and the absence of any other fibroid tumours in the uterine walls.

Dr. Salisbury's third specimen was a uterus from a married woman, aged sixty years, who gave a history of offensive vaginal hæmorrhage of short duration. The climacteric had occurred at the age of forty-five years, and she had been symptom-free until four weeks prior to operative treatment. On examination of the patient the uterus was small, smooth and freely mobile, and the cervix appeared healthy. During curettage a large amount of friable necrotic tissue was removed from the cavity of the uterus, and this had the macroscopic appearance of neoplastic tissue. Total hysterectomy and bilateral salpingo-oopherectomy were carried out. Examination of the specimen revealed an extensive friable carcinoma of the body of the uterus, and microscopic examination of sections showed that the muscular layer of the uterus had been invaded almost to the peritoneal layer. Dr. Salisbury said that the point of interest in this case was the complete absence of any signs and symptoms from such an advanced carcinoma. Frequently carcinoma of the body of the uterus was a difficult condition to diagnose in the early stage, owing to the absence of signs and symptoms. The importance of diagnostic curettage in all cases of post-menopausal hæmorrhage was stressed.

Endometriosis.

DR. K. S. RICHARDSON showed a female patient, aged twenty-eight years, married, but with no children, and having had no miscarriages, who had suffered from pain in the lower part of the abdomen since the age of sixteen years. The pain was gradually becoming worse; it was aching in character and was not accompanied by nausea or vomiting. The pain was much more severe with the menstrual periods, both before and during menstruation; it then radiated to the legs. Slight pain was felt on defæcation, accentuated during the menses. The menarche had occurred at the age of fifteen years; the menstrual periods had at first been irregular, becoming regular at the age of eighteen years. The cycle was of the twenty-eight day type, the periods lasting for six days, and the loss was of average amount. In attempts to relieve the pain she had undergone the following procedures: (i) appendicectomy at Saint George Hospital eleven years previously; (ii) manipulation at Saint George Hospital nine years previously; (iii) an Alexander Adams operation for external shortening of the round ligaments five years previously. In 1946 she had presented signs and symptoms of hyperthyreoidism, the diagnosis being confirmed by estimation of the basal metabolic rate, which was 18%, and thiouracil treatment was successful.

Dr. Richardson said that he had examined the patient in June, 1947, on account of the persistent pain. Some generalized tenderness without rigidity was present in the lower part of the abdomen. Vaginal examination showed the uterus to be small and anterior in position; the left fornix was normal and the cervix appeared normal on inspection. In the right fornix some hardness was present, which extended into the posterior fornix, where there was a small, nodular, hard mass; it was fixed and tender and seemed to be attached to the rectum behind and to the cervix anteriorly. No other abnormalities were detected on physical examination. A provisional diagnosis of pelvic endometriosis was made, and operation was recommended.

On July 18 curettage and section were performed. The previous pelvic findings were confirmed under anæsthesia. Microscopic examination of the endometrium revealed an early secretory phase. On section a mass was found in the cul-de-sac about the size of a large walnut, with dense adhesions forward to the cervix and backward to the rectum. The ovaries were not involved in the mass except for some light adhesions, which were easily severed. No other abnormality was detected. Dr. Richardson said that he considered that surgical removal of the mass was impossible without the risk of radical pelvic surgical measures, and as the function of child-bearing had to be conserved the abdomen was closed. It then became necessary to look for some form of treatment which might alleviate the symptoms, and it was decided to give progesterone therapy a trial. This decision was made on the basis of the theory propounded by Goodall, of Montreal, that endometriosis was a manifestation of hyperœstrinism, and that the frequently associated sterility was due to this hormonal imbalance. The patient was given progesterone in the form of "Lipo-Lutin" (Parke, Davis and Company), five milligrammes by injection three times a week during her stay in hospital (three weeks), and was then discharged from hospital. She said that the pain was much relieved, and that a menstrual period which had occurred during her last week in hospital had been much less painful than before. On her discharge from hospital she was given progesterone in a form to be taken orally ("Ethisterone") for one month. She had reported one week prior to the meeting. While taking progesterone she had had a painless menstrual period, but after the treatment was discontinued the next period was somewhat painful. For the past month she had taken one tablet a day and thought that the pain was controlled. Pelvic examination revealed the presence of the mass, which had diminished in size and was not so tender as previously. Dr. Richardson said that he had suggested that she should have a course of three months, taking one tablet per day for the fourteen premenstrual days, and that he intended to review the position again at the end of that time.

Dr. Richardson next showed an unmarried female patient, aged forty-four years, who complained of dysmenorrhœa of thirteen years' duration. The pain occurred before and during menstruation and was felt in the mid-line. The menarche had occurred at the age of thirteen years and the menses had always been regular, occurring every twenty-

eight days and lasting for five days. Over the last five years the amount lost had been increasing. During the past six months a small but constant intermenstrual discharge had been present. The patient also complained of generalized lower abdominal discomfort.

Examination showed the patient to be an obese woman with some generalized lower abdominal tenderness, no palpable masses and no abnormality in any of the general physical systems. Vaginal examination was difficult owing to the obesity and the virginal introitus, and it was decided to make an examination under anæsthesia. This was done, and a uterus of about normal size was palpated. The passage of a sound was difficult, owing to the presence of what was thought to be a submucous fibroid tumour. In the lateral fornices were masses about the size of an egg, firm, tender to touch and seemingly adherent to the uterus and the lateral pelvic walls. Operation was performed and the masses were found to be chocolate cysts. The uterus was not large, and it was found that the tubo-ovarian masses could be dissected free. Double salpingo-oophorectomy was performed. Recovery was uneventful. Three months after operation the patient was well, free from pain and leading an active life.

Complete Prolapse following Total Hysterectomy.

Dr. Richardson then showed a female patient, aged fifty-nine years, who had had five children. She had been troubled for years with a sensation of something falling. She had frequency of micturition, but no stress incontinence. The lump became more troublesome during the latter part of the day, and could be pushed back when she was lying down. The bowels were costive. She had been badly torn during the first labour, but the other four had been uncomplicated. She had undergone a thyreoidectomy eight years prior to the meeting. A total hysterectomy had been performed twenty-one years previously on account of hæmorrhage. The sensation of a lump was present when the hysterectomy was performed, but during the last ten years it had become much worse. The patient suffered from headache and shortness of breath. Physical examination revealed no general abnormality except raised blood pressure (210 millimetres of mercury, systolic, and 110 millimetres, diastolic). Vaginal examination revealed a lax introitus with pronounced cystocele and rectocele; complete prolapse of the vaginal vault occurred when the patient coughed, the outlet being turned into a completely inverted dome. At the summit of the dome was a slightly puckered line, indicating the line of suture of the vaginal vault.

Under ether anæsthesia the area of puckering was seized with a volsellum and drawn down, and a longitudinal incision was made in the anterior vaginal wall through to the vesico-vaginal fascia. The bladder was dissected free and pushed well up. Undersewing of the vesico-vaginal fascia was carried to within half an inch of the puckered line, and this was reinforced by buttress sutures (mattress) passing through the vaginal mucosa and fascia; the excess was then cut away and a thin catgut suture completed the anterior colporrhaphy. Similarly a posterior colpoperineorrhaphy was performed with adequate apposition and suture of the *levatores ani*. The remaining vaginal mucosa still attached to the summit of the dome was now dissected free from the vesico-vaginal and recto-vaginal fascia, and through these layers a series of two purse-string sutures was placed, the capacity of the vault being thus diminished. When these sutures were tied the vault was found to be well held up, and the remaining edges of the mucosa were apposed.

The post-operative course was reasonably satisfactory. The patient's bladder was catheterized for two days, and on the third day she passed urine well. On the fifth day some sloughing of the anterior suture line occurred; however, it did not break down. The perineum broke down, and on the fourteenth day after operation it was resutured under local anæsthesia. In all the patient was in hospital for five weeks, and the end result was satisfactory. During all the post-operative period she was given nine milligrammes of stilbœstrol per day with the object of increasing the vascularity of the parts. When examined three weeks before the meeting (two and a half months after operation) the patient was symptom-free, the vault was in good position, and no cystocele or rectocele was present.

Dr. Richardson said that the condition was rare, and had two general causes: (i) its presence before hysterectomy, and the failure to recognize and treat it before ablation of the uterus; (ii) the failure to utilize properly the ligamentous supports in rebuilding the pelvic floor. It was important that, in the performance of total hysterectomy, the parametrium on each side should be attached to the round ligament either by suture or by firm tying, so that the pelvic floor was thus slung and attached to the pelvic side walls. The condition was less common after subtotal hysterectomy, as in that operation the amputation was usually performed above the level of the cardinal ligaments. The type of operation performed in the case under discussion had been chosen because the essential point to recognize was the presence of the cystocele and rectocele. The repair of these could not be achieved except by standard methods, and it was thought that any intraabdominal attempt to sling the vault would be unsatisfactory. Such an operation would be justifiable only in the case of a young women, in which the preservation of a normal calibre and length of vagina was considered necessary. Such operations had been described by Grant Ward (*Archives of Surgery*, Volume XXXVI, 1938, page 163) and by Norman Miller (*Surgery, Gynecology and Obstetrics*, Volume XLIV, 1927, page 550).

Hæmatocolpos.

DR. M. M. KENNEDY showed a girl, aged sixteen years, who had been operated on for hæmatocolpos. The history was strongly suggestive of a traumatic cause when the child was aged four years. Dr. Kennedy said that if that was so, it was the only such case he had found in the literature. The alternative possibility of a congenital origin of the vaginal stricture was discussed.

Adenomyoma.

Dr. Kennedy's second patient was a woman, aged forty-two years, who had undergone total hysterectomy on account of adenomyoma. The main interest lay in the pre-operative diagnosis, which had proved difficult, and which had been thought to be incomplete miscarriage or fibromyoma. At operation a large tumour was found to have practically replaced the posterior wall of the uterus. Dr. Kennedy showed the histological slide in which myoma was seen to be present.

Incarcerated Retroverted Gravid Uterus.

DR. A. M. MACINTOSH showed a married woman, aged thirty-five years, who had been admitted to hospital on May 15, 1947, complaining of severe abdominal pain of three days' duration, of inability to pass urine for three days and of "dribbling" of urine during that period. The last menstrual period had occurred on March 13, and the patient had had some difficulty with her bowels. The menarche had occurred at the age of thirteen years. The menstrual periods occurred on a twenty-eight to thirty day cycle and lasted for five or six days. The patient had two children, the younger aged sixteen years.

Examination of the patient revealed a large, tense, abdominal tumour. Vaginal examination revealed a large, tender mass in the pelvis; the cervix was high up under the symphysis and dilated to admit one finger.

An indwelling catheter was inserted into the bladder and fifty ounces of urine were slowly withdrawn, with the result that the abdominal tumour disappeared and symptoms were much relieved. On May 16 the patient was examined under anæsthesia, and a provisional diagnosis was made of (i) incarcerated retroverted gravid uterus or (ii) pregnancy with incarcerated ovarian cyst or fibroid tumour. It was impossible to push the mass in the pelvis over the brim, and after consultation with Dr. K. S. Richardson and Dr. C. V. Salisbury it was decided to adopt conservative measures, leaving the catheter *in situ* and keeping the bowels open. Vaginal examination on May 18 showed the pelvic mass to be slightly more movable. Slight vaginal hæmorrhage occurred. A vaginal examination on May 20 showed that the mass was obviously rising out of the pelvis. A slight amount of dark blood was discharged for several days afterwards. Examination on May 27 showed the mass to be a pregnant uterus well up in the abdomen; pronounced sacculation of the posterior wall was still present. On November 24 the patient was delivered of a full-time female infant.

Dr. Macintosh said that the first problem was of diagnosis—whether the condition was a pregnancy only or a pregnancy associated with a cyst or a fibroid tumour. The next problem was the question of watchful expectancy or active interference. After much thought the course adopted had been decided upon.

Chocolate Cyst, Intramural Fibroid Tumour, Endometriosis.

Dr. Macintosh next showed an unmarried female patient, aged thirty-three years, who had been admitted to hospital on July 17, 1947, with the provisional diagnosis of appendicitis

and ovarian cyst. The patient had been complaining of attacks of pain in the right side for the past five months. The first attack had been accompanied by vomiting, and the pain was severe, doubling her up. The last attack had occurred two days previously and was still present. The menarche had occurred at the age of thirteen years. The menstrual periods occurred every twenty-five days and lasted for five or six days; no menorrhagia had occurred. She suffered from severe dysmenorrhœa before, during and at the end of the menstrual periods. She had had no previous illnesses.

On examination the patient was seen to be a woman who looked older than her stated years; hirsuties was present on the upper lip and chin. She was nervous and excited; her pulse rate was 120 per minute. The abdomen was obese. A large, firm, oval tumour was visible and palpable in the hypogastrium; it was ballottable and cystic and not attached to the abdominal wall. Vaginal examination showed that the patient was *virgo intacta*. Rectal examination revealed no definite abnormalities, but the mass felt hard and the uterus could not be made out.

On July 18, under "open" ether anæsthesia, subtotal hysterectomy and double salpingo-oophorectomy were carried out. In the right ovary a large chocolate cyst about six inches in diameter was found. The uterus, bowel and pouch of Douglas were affected by endometriosis. The left ovary was cystic and the uterus was hard and enlarged. The large cyst was firmly adherent to the posterior pelvic wall and ruptured during removal.

The body of the uterus and both ovaries were sent for pathological examination. The uterus measured three and a half inches by three inches by three inches and contained a large intramural fibroid tumour. One ovary had been replaced by a large, thick-walled cyst with a smooth lining, measuring three or four inches in diameter. The other ovary contained several cysts. The two largest of these measured one inch in diameter. One of these ruptured, and the other contained mucoid material. Microscopic examination of sections from each ovary confirmed the clinical diagnosis of endometriosis.

Multiple Urinary Calculi.

DR. R. I. CAMPBELL showed a female patient, aged seventy-five years, who had been admitted to hospital in the care of Dr. A. C. Thomas on July 24, 1946. She complained of not feeling well, of being tired, of loss of appetite and of vomiting of about seven weeks' duration. She had noticed a lump in the right side of the back for about a month. She had no urinary symptoms, except that she had to pass urine two or three times at night.

On examination of the patient, a hard irregular mass was felt in the right side of the abdomen. It was slightly mobile, but not tender. In the right side of the back there was a smooth, fairly superficial, irregular swelling. It was soft and fluctuant and tender, and the skin around it was œdematous. Her temperature was 101·4° F. and her pulse rate 120 per minute. On July 27 a blood count revealed a leucocytosis of 15,600 per cubic millimetre. On July 29 an X-ray examination revealed a huge collection of stones on the right side extending from the level of the upper border of the third lumbar vertebral body to well over the sacrum, with a linear arrangement extending into the right side of the pelvis; it also revealed a soft-tissue swelling on the right side posteriorly. On July 31 her blood urea level was 80 milligrammes per 100 millilitres, and urine examination showed pus and red cells to be abundant.

On August 8 a large abscess in the right loin was opened and drained. There was a copious discharge from this wound for about a fortnight, and the discharge continued until her next operation on August 29. On August 24 her blood urea level was 28 milligrammes per 100 millilitres.

On August 29 an extraperitoneal nephrectomy and partial ureterectomy were performed through a right oblique abdominal incision, extending into the right loin. A huge, grossly disorganized kidney, portion of a greatly dilated ureter and a huge number of calculi of varying sizes were removed. After this second operation she was given 15,000 units of penicillin every three hours until September 5, when she had had a total of 855,000 units. Her convalescence was uneventful, her wounds healed quickly, and she was discharged from hospital on September 26 feeling well.

The foregoing operations were performed by Dr. A. C. Thomas.

Dr. Campbell said that on November 8, 1946 the patient was again admitted to hospital under his care, complaining of pain in the abdomen and left loin, and of not having passed urine for three days. She had had some intermittent vomiting. On examination of the patient, tenderness was present in the left loin and over the left lower quadrant of the abdomen. Her tongue was dry and furred. Catheterization produced only one ounce of clear urine. Sodium sulphate solution was given intravenously, but no urine was passed. On November 9 an X-ray examination revealed a small shadow, probably a calculus, close to the tip of the fifth lumbar transverse process on the left side. Some calculi were also present in the stump of the right ureter.

On November 10 there was no improvement in the patient's condition, and she had passed no more urine. An attempt to pass a catheter up the left ureter was not successful, so operation was decided on. A uretero-lithotomy was performed through a lumbar incision, and a small calculus was found impacted in the ureter, about two inches below the renal pelvis. This was removed, and the wound was closed with a small drainage tube down to the ureter. She passed 62 ounces of urine during the next twenty-four hours, and then made rapid progress towards recovery; she was discharged from hospital on December 9 feeling well.

Dr. Campbell said that there were many interesting features about the case. The patient had had an extraordinary number of stones in a grossly enlarged and disorganized kidney and ureter, which she had undoubtedly had for many years. She was seventy-five years of age, and her recovery after each operation was rapid and uneventful. She had had anuria for almost five days before her last operation.

Carcinoma of the Bladder.

Dr. Campbell next showed a male patient, aged sixty-four years, who had been admitted to hospital on February 10, 1947, with a history of hæmaturia occurring for the first time two weeks previously. He had also had some scalding with micturition, but only slight frequency, passing urine six times during the day and twice during the night. He had no other urinary symptoms. The attack had cleared up after twenty-four hours, but had recurred on the day of his admission to hospital.

On examination of the patient the prostate was found to be only slightly enlarged. Urine examination revealed a heavy deposit of pus cells and a number of red cells. X-ray examination revealed no calculi. On February 22 a cystoscopic examination was performed, and this revealed a tumour, about the size of a small cherry, just behind the right ureteric orifice. Its pedicle was sessile, and there appeared to be some infiltration of the surrounding bladder wall. Its villi were stunted and swollen. The tumour was undoubtedly malignant.

On March 8 the bladder was opened, and the tumour was excised by diathermy together with a good margin of bladder wall. The bladder was closed around a White's suprapubic tube. The patient's convalescence was rather stormy, as he developed bronchopneumonia which took a considerable time to clear up. However, he was finally discharged from hospital on May 14. He had been free of any urinary symptoms for several weeks before his discharge. He was told to report for cystoscopy again in about four months' time.

On September 13 cystoscopy was carried out again. The bladder was free of any tumour, and there was no evidence of cystitis. Dr. Campbell said that he had shown the patient to demonstrate the value of early cystoscopy in cases of hæmaturia. The patient had reported and been examined shortly after his first attack of hæmaturia, and as a result an early carcinoma was found and treated by local excision, with so far gratfying results. It was proposed to keep him under observation by cystoscopic examinations at regular intervals.

Foreign Body in the Urethra.

Dr. Campbell finally showed a male patient, aged thirteen years, who had been admitted to hospital on June 6, 1947, complaining of hæmorrhage from the urethra occurring intermittently for about a month. The blood would appear at any time, and not with micturition. He did not complain of frequency or of painful micturition. On examination of the patient something hard could be felt in the mid-line in the perineum, and an X-ray examination showed this to be a needle in the posterior portion of the urethra. On further questioning of the patient, he admitted that he had put the needle in the urethra himself, and that he had been unable to get it out.

On June 7 an external urethrotomy was performed, the urethra being incised over the end of the needle. The needle, which was a large darning needle, was then easily removed. The urethra was then sutured, the perineal wound was

completely closed, and a rubber catheter was tied into the bladder. The indwelling catheter was left for three days, at the end of which time the patient passed urine without trouble. He was discharged from hospital on the eleventh day.

(To be continued.)

Medical Societies.

THE MEDICAL WOMEN'S SOCIETY OF NEW SOUTH WALES.

THE annual meeting of the Medical Women's Society of New South Wales was held at the Secondary Schools' Club, Gowing's Building, Market Street, Sydney, on March 31, 1948, Dr. Grace Cuthbert, the President, in the chair.

Before proceeding to the business of the meeting, the President welcomed Mesdames Hand, Brown and Ford from New Zealand and Dr. M. Heseltine on her return from the United States of America. New members nominated for election to the society were also welcomed by the President.

The President paid tribute to the late Dr. Edna Nelson, whom she described as a brilliant colleague and public-spirited woman. A tribute was also paid to the late Mrs. Benjamin Edye. The sympathy of the society had been expressed to the families of both.

Minutes.

The minutes of the previous annual meeting were read and confirmed.

Congratulations.

The President congratulated Dr. M. Hamilton on her election as a representative of the Medical Women on the Council of the New South Wales Branch of the British Medical Association and Dr. L. Beveridge as an ordinary member of the Council.

Annual Report.

The annual report of the Secretary was read and received. The report is as follows.

The membership of the society now stands at 140. Seventeen new members were elected during the year and one resigned. There were four general meetings held during the year, the average attendance being 31—not quite as good as in 1946.

At the annual meeting Dr. Jeanette Robinson gave a most interesting account of her experiences in a Japanese prisoner-of-war camp. At the June meeting Mrs. J. Cardno and Miss Kate Ogilvie gave short addresses on "The Functions of the Social Worker in the Community" and "The Function of the Medical Social Worker". Both talks were most instructive for hospital workers and private practitioners.

The clinical meeting for 1947 was held at the Renwick Hospital for Infants, Summer Hill, in October. Special thanks are due to Dr. Dalgarno, Dr. Mona Nelson and Dr. S. C. Puckey and staff of the Renwick Hospital for a most interesting and enjoyable afternoon.

At the fourth meeting Dr. I. B. Saunders gave a brief account of her trip to the United States of America and Dr. Nell McMahon gave a short outline of her work in England.

Committee Meetings.

The committee held five ordinary meetings and two special meetings. Important matters dealt with included: (i) Salaries of medical women in the State Public Service. (ii) Appointment of medical women to teaching hospitals. (iii) Affiliation of the Australian Federation of Medical Women with the Australian Liaison Committee of national women's organizations. (iv) Prize for medical women to be awarded annually by the Medical Women's Society of New South Wales for a period of five years. (v) Nomination of a woman representative to the Council of the New South Wales Branch of the British Medical Association. (vi) The resignation of the President, Dr. Lucy Bryce, and the Secretary-Treasurer, Lady MacKenzie, of the Australian Federation of Medical Women, and the temporary appointment of the President and Secretary-Treasurer of the Medical Women's Society of New South Wales to office until the Perth Conference in 1948, when it is hoped sufficient women will be present to elect office-bearers according to the constitution of the Australian Federation of Medical Women.

Representation.

Dr. Cuthbert, as President of the Medical Women's Society of New South Wales, was nominated to represent the Australian Federation of Medical Women on the Australian Liaison Committee. It was proposed by Dr. M. Hamilton and seconded by Dr. Dalgarno and carried that Dr. Cuthbert and Dr. M. C. Puckey should continue to represent the society in matters affecting the salaries of medical women in the State Public Service.

General.

The report of the subcommittee consisting of Dr. Cuthbert, Dr. Anderson, Dr. Cuningham and Dr. M. C. Puckey that the prize for the Medical Women's Society should be awarded annually for a period of five years was adopted.

A donation of £10 10s. was made to the Margaret Harper Testimonial Fund, and members decided to support the Florence Nightingale Scholarship Fund, a sum of £24 being collected to date.

The usual graduands' party was not held during the year because of the lateness of the final examinations. In February the President entertained the committee and new members at a most enjoyable late afternoon party.

The committee wish to thank the President for permitting meetings to be held at her flat and for the numerous times she provided supper. It is also grateful to the Girls' Secondary Schools Club for accommodation for the general meeting.

Finance.

The balance sheet for the year shows a satisfactory position. The committee wishes to express its appreciation of the service of the honorary auditors, Dr. Marjorie Little and Dr. Elyse Dalyell.

Society's Address.

The permanent address for the Medical Women's Society of New South Wales is to be the Rachel Forster Hospital, Redfern.

Election of Office-Bearers.

The following office-bearers for 1949 were elected unanimously: *President*: Dr. Grace J. Cuthbert; *Vice-Presidents*: Dr. P. Anderson and Dr. K. Cuningham; *Honorary Secretary*: Dr. D. Rutherford; *Honorary Treasurer*: Dr. L. Sharfstein; *Committee Members*: Dr. M. C. Puckey, Dr. P. Kilburn, Dr. Burton-Bradley, Dr. M. Lane, Dr. L. Pulstone-Jones, Dr. B. Manuel, Dr. H. Moore.

New Members.

The following new members were unanimously elected: Dr. J. K. Williams, Dr. J. J. Turner, Dr. M. Thomson, Dr. S. Repin, Dr. J. Bowen, Dr. L. Benson, Dr. A. Robey and Dr. P. Braithwaite.

Guest Speaker.

At the conclusion of the business of the meeting Madame Rambert of the Ballet Rambert gave a most interesting talk on the ballet which was greatly enjoyed.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-End Course at Lismore.

THE Post-Graduate Committee in Medicine in the University of Sydney announces that a week-end course will be held at the Lismore Municipal Council Chambers, Lismore, in conjunction with the North-Eastern Medical Association on Saturday and Sunday, August 7 and 8, 1948. The programme will be as follows.

Saturday, August 7: 2.15 p.m., "Role of Cæsarean Section", Dr. John Chesterman; 4.15 p.m., "Gynæcological Pathology", Dr. Mary Heseltine; 8 p.m., "Termites, Totalitarianism and You", Dr. John Bostock.

Sunday, August 8: 9.30 a.m., "Laboratory Investigations in Gynæcology", Dr. Mary Heseltine; 11.15 a.m., "Some Obstetrical and Gynæcological Problems", Dr. John Chesterman; 2 p.m., meeting of the North-Eastern Medical Association.

The fee for the course will be £2 2s. Those wishing to attend are requested to notify Dr. Nugent Brand, Honorary Secretary, North-Eastern Medical Association, 39, Conway Street, Lismore, as soon as possible.

Course in Gynæcology and Obstetrics.

The Post-Graduate Committee in Medicine in the University of Sydney announces that in conjunction with the honorary medical staff of the Royal Prince Alfred Hospital a course in gynæcology and obstetrics will be held at the King George V Memorial Hospital for Mothers and Babies, Camperdown, for two weeks from August 30 to September 10, 1948. The fee for the course will be £5 5s., and registration is limited to twelve (12) persons. Applications for enrolment, accompanied by a remittance for the amount of fee, should be made to the Course Secretary, the Post-Graduate Committee in Medicine, 131, Macquarie Street, Sydney, as soon as possible. Telephones: B 6980-BW 7483. Telegraphic address: "Postgrad, Sydney." The programme will be as follows.

Gynæcology.

Monday, August 30: 9.30 a.m., "Perineal Laceration and Repair", Dr. F. A. Maguire; 11 a.m., Operative Demonstration, "Perineorrhaphy", Dr. F. A. Maguire; 12 noon, Question Session, Dr. F. A. Maguire; 2 p.m., "Carcinoma of the Corpus Uteri", Dr. F. N. Chenhall; 3.30 p.m., "Ectopic and Other Sites of Nidation", Dr. M. J. L. Stening.

Tuesday, August 31: 9.30 a.m., "Impressions from Abroad", Dr. G. G. L. Stening; 11 a.m., "Endocrines in Gynæcology", Dr. E. M. Day; 12 noon, Question Session, Dr. G. G. L. Stening, Dr. M. J. L. Stening, Dr. E. M. Day; 2 p.m., Operative Demonstration, Dr. F. N. Chenhall; 3.30 p.m., "Cervical Infection", Dr. Muriel McIlrath.

Wednesday, September 1: 9.30 a.m.: "Surgery of Prolapse" (colour film), Dr. H. H. Schlink; 11 a.m., Operative Demonstration, "Manchester Repair", Dr. H. H. Schlink; 2 p.m., "Ovarian Tumours", Dr. M. B. Fraser; 3.30 p.m., "Recent Investigations in Gynæcological Pathology", Dr. Mary Heseltine.

Thursday, September 2: 9.30 a.m., "Carcinoma of the Cervix Uteri", Dr. G. G. L. Stening; 11 a.m., "Disorders of Menstruation", Dr. C. L. Chapman; 12 noon, Question Session, Dr. C. L. Chapman, Dr. M. B. Fraser, Dr. J. C. Loxton; 2 p.m., Operative Demonstration, Dr. G. G. L. Stening; 3.30 p.m., Demonstration of Cases, Dr. Margaret Mulvey.

Friday, September 3: 9.30 a.m., "Gynæcological Pathology", Dr. Mary Heseltine; 11 a.m., "Sterility", Dr. F. N. Chenhall; 12 noon, Question Session, Dr. F. N. Chenhall, Dr. Mary Heseltine, Dr. M. Mulvey; 2 p.m., "Preservation of Function in Gynæcological Surgery", Dr. J. C. Loxton; 3.30 p.m., "Endometriosis", Dr. C. L. Chapman.

Obstetrics.

Monday, September 6: 9.30 a.m., "Obstetrical Pathology", Dr. Mary Heseltine; 11 a.m., "Toxæmia of Pregnancy", Professor B. T. Mayes; 12 noon, Question Session, Professor B. T. Mayes, Dr. R. C. Gill, Dr. R. F. Back, Dr. Mary Heseltine; 2 p.m., "Pregnancy following Eclampsia", Dr. R. F. Back; 3.30 p.m., "Cæsarean Section" (film or operative demonstration), Professor B. T. Mayes.

Tuesday, September 7: 9.30 a.m., "Endocrines in Obstetrics", Dr. R. C. Gill; 11 a.m., "Vomiting in Pregnancy", Dr. A. F. Hobson; 2 p.m., "Signs of Disease in the Newborn", Dr. N. C. Cunningham; 3.30 p.m., "Head Injuries in the Newborn", Dr. S. P. Bellmaine.

Wednesday, September 8: 9.30 a.m., "Ante-Partum Hæmorrhage", Dr. H. Bruce Williams; 11 a.m., "Bleeding in the First Trimester", Dr. F. P. Piggott; "Anuria in Obstetrics", Dr. J. M. Farrar; 2 p.m., "Manual Removal of Placenta", Dr. W. D. Cunningham; 3.30 p.m., "Breech Delivery" (colour film), Professor B. T. Mayes.

Thursday, September 9: 9.30 a.m., "Minor Obstetrical Problems", Dr. M. McAuley White; 11 a.m., Ward Rounds, Dr. H. Bruce Williams; 12 noon, Question Session, Dr. H. B. Williams, Dr. A. F. Hobson, Dr. McAuley White; 2 p.m., "The Lacerated Perineum in Obstetrics", Dr. Elton Holman; 3.30 p.m., "Heart Disease in Pregnancy", Dr. R. F. Back.

Friday, September 10: 9.30 a.m., "Surgical Complications of Pregnancy", Dr. W. D. Cunningham; 11 a.m., "Anæsthesia in Obstetrics", Dr. W. I. T. Hotten; 12 noon, Question Session, Dr. W. D. Cunningham, Dr. W. I. T. Hotten, Dr. Elton Holman; 2 p.m., "Radiology of the Female Pelvis", Dr. D. G. Maitland; 3.30 p.m., "Puerperal Sepsis", Professor B. T. Mayes.

THE MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

General Practitioners' Refresher Course.

Monday, September 6, at the Royal Melbourne Hospital: 10 a.m., "Surgery of the Gall-Bladder", Dr. G. R. A. Syme; 2 p.m., "Pneumonia: Diagnosis and Management", Dr. L. Hurley.

Tuesday, September 7, at the Alfred Hospital: 10 a.m., a surgical subject, Dr. R. Officer; 2 p.m., "Bacterial Endocarditis", Dr. E. Clarke.

Wednesday, September 8, at Saint Vincent's Hospital: 10 a.m., "The Problem of the Stiff Joint", Dr. G. Shaw; 2 p.m., "Cardiological Problems in General Practice", Dr. J. G. Hayden.

Thursday, September 9, at the Children's Hospital: 10 a.m., "Common Surgical Ailments in Children", Dr. R. Howard; 2 p.m., "Some Common Clinical Problems in Pædiatrics", Dr. V. L. Collins.

Friday, September 10, at the Eye and Ear Hospital: 10 a.m., "The General Practitioner's Consideration and Management of Deafness", Dr. Jean Littlejohn; 2 p.m., "Eye Conditions in General Practice", Dr. A. Anderson.

Saturday, September 11, at the Medical Society Hall: 10 a.m., "Legal and Ethical Problems in Medical Practice", Dr. C. H. Dickson.

Monday, September 13, at the Royal Melbourne Hospital: 10 a.m., "Hernia", Dr. D. Leslie; 2 p.m., "Congestive Cardiac Failure", Dr. K. Grice.

Tuesday, September 14, at the Alfred Hospital: 10 a.m., "Scrotal Swellings", Dr. C. A. M. Renou; 2 p.m., "Diagnosis and Treatment of Peptic Ulceration", Dr. R. Andrew.

Wednesday, September 15, at Saint Vincent's Hospital: 10 a.m., a urological subject, Dr. H. Mortensen; 2 p.m., "Cases of Neurological Interest", Dr. J. Billings.

Thursday, September 16, at Prince Henry Hospital: 10 a.m., "Common Disorders of the Peripheral Blood Vessels", Dr. S. Reid; 2 p.m., "Nephritis", Dr. P. S. Woodruff.

Friday, September 17, at the Royal Park Hospital: 10 a.m., "Psychiatric Emergencies", Dr. J. K. Adey; 2 p.m., at the Infectious Diseases Hospital, Fairfield: "The Diagnosis and Management of the Common Infectious Diseases", Dr. J. McLorinan.

In addition to the subjects arranged, opportunity will be given for discussion of problems submitted by those attending the course. Where possible arrangements will be made for lunch at the hospital where the morning session is held. Those wishing to avail themselves of this facility should notify the hospital before 10.30 a.m. The fee for this course is £10 10s., and the class will be limited to 20.

This course coincides with a series of evening lectures given by Professor J. C. Spence, M.D., F.R.C.P., Nuffield Professor of Child Health, the Royal Victoria Infirmary, Newcastle, England.

Application for enrolment should be made a fortnight before the commencement of the course to the Secretary of the Committee, 426, Albert Street, East Melbourne. Telephone: JM 1547.

Correspondence.

THE PHARMACEUTICAL BENEFITS ACT.

SIR: There has recently appeared a political pamphlet by Senator the Honourable N. E. McKenna, Minister for Health and Social Services, on the pharmaceutical benefits proposals. It would be an understatement to call this diatribe anything less than a magnificent school of red herrings. From the Senator's very fine collection I select for comment two of particularly vivid hue. One is his pathetic appeal: "I say to the doctors of Australia that they get their living—some get their wealth and a few get their very great wealth—from the sick and suffering in the community." Maybe we do, but only in the same sense that producers of foodstuffs get their living from the starving,

clothiers get theirs from the naked, and lawyers get theirs from the criminals—murderers, thieves, prostitutes and such like—a pretty state of affairs. The other is a more serious point. In speaking of the Federal Council's list of essential drugs suggested for a free list, the Minister mentions sulpha drugs, penicillin, streptomycin and a few others, and goes on to say: "These are the common ailments which would not be touched in any way if we were to adopt the limited list suggested by the Federal Council of the British Medical Association: coughs, tonsillitis, constipation, conjunctivitis, vomiting, tuberculosis, *angina pectoris*, threadworm, rheumatism in its various forms, skin diseases (such as ringworm), gout, epilepsy, sciatica, anæmias (except pernicious anæmia), bronchitis, influenza, indigestion, bladder affections, gastric and duodenal ulcers, measles, goitre, headaches, migraine, lumbago, sleeplessness, catarrh in its various forms, kidney complaints, burns and scalds, chicken-pox, pleurisy, neuralgia, neurosis and neurasthenia, fibrositis and abrasions." That a politician should make such an obviously inaccurate claim is perhaps not novel. What is really disconcerting here is that this is an authoritative ministerial statement. Whether this confused medley of symptoms and diseases is the Minister's own invention or was supplied to him by those whom he believes to be "experts of the highest levels" is immaterial. The Minister is satisfied to accept and publish this loose and grossly inaccurate statement as fact. Such are the foundations that the pharmaceutical benefits scheme is to be built on.

Yours, etc.,

O. E. NICHTERLEIN.

Wilpena Street,
Eden Hills,
South Australia.
July 15, 1948.

PENICILLIN.

SIR: It seems to be generally recognized in England that the pure white crystalline penicillin is a great advance on the yellow penicillin. I feel that the Commonwealth Serum Laboratories should make this product available in Australia. I have seen the crystalline penicillin in solution used in injections into the aqueous and vitreous of the eye to combat infection. It is everywhere used subconjunctivally. The pure white crystals are also placed in wounds. I have yet to see the yellow penicillin to be used in England and I believe it is now superseded by the pure product.

Yours, etc.,

F. W. SIMPSON.

Nuffield Laboratory,
Eye Hospital,
Oxford,
England.
May 28, 1948.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 91, 97 and 104, of June 10 and 24 and July 8, 1948.

PERMANENT NAVAL FORCES OF THE COMMONWEALTH (SEA-GOING FORCES).

Appointment.—Bertram Charles Morgan is appointed Surgeon Lieutenant (for Short Service), dated 1st October, 1948.

AUSTRALIAN MILITARY FORCES.

Interim Army.

Australian Army Medical Corps.

NX206866 Captain R. Wilkinson is transferred to the Reserve of Officers (Australian Army Medical Corps) (2nd Military District), 10th March, 1948.

116th Convalescent Depot: To be Temporary Major, 23rd February, 1948.—NX502739 Captain C. Radeski.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

The appointment of Flight Lieutenant M. Cohen (297484) is terminated on demobilization, 29th April, 1948.

The part-time appointment of Temporary Squadron Leader L. H. Albiston (256799) is terminated on cessation of part-time duties, 13th May, 1948.

Reserve: Medical Branch.

The following ex-officers are appointed to commissions with ranks as indicated: Temporary Squadron Leader Leslie Howard Albiston (256799), 14th May, 1948, Flight Lieutenant Howard Stewart Moore (266931), 1st May, 1948.

The Royal Australasian College of Physicians.

Meeting of the College.

THE ordinary meeting of the Royal Australasian College of Physicians in 1948 will be held at Melbourne on Thursday, Friday and Saturday, October 21, 22 and 23, 1948.

Examination for Membership.

An examination for membership of the Royal Australasian College of Physicians will take place in September-October, 1948. The written examination will be held in capital cities of the Commonwealth where candidates are offering. The clinical examination will take place at Melbourne. Only those candidates whose answers in the written examination have attained a standard satisfactory to the Board of Censors will be allowed to proceed to the clinical examination. The written examination will be conducted in capital cities on Saturday, September 18, 1948. The clinical examination will be conducted at Melbourne on Monday, Tuesday and Wednesday, October 18, 19 and 20, 1948. Applications to appear before the Board of Censors should be made in the prescribed form and must be in the hands of the honorary secretary of the College not later than Saturday, August 21, 1948. Application forms are obtainable from the Honorary Secretary, 145, Macquarie Street, Sydney.

The Royal Australasian College of Surgeons.

THE next meeting of the Court of Examiners for the primary examination for Fellowship of the Royal Australasian College of Surgeons will be held in Melbourne, probably beginning on Monday, November 22, 1948. Candidates who desire to present themselves at this meeting should apply to the Censor-in-Chief for permission to do so on or before October 1, 1948. The appropriate forms are available from the Secretary of the Royal Australasian College of Surgeons, Spring Street, Melbourne.

Obituary.

DAVID GIFFORD CROLL.

WE are indebted to Dr. E. S. Meyers for the following appreciation of the late Dr. David Gifford Croll.

My first recollection of Croll was in the year 1909 when somebody pointed him out to me outside the orderly room of the Sydney University Scouts, situated in those days behind the Macleay Museum.

Most of my close associations with Croll sprang from a common interest in the affairs of the British Medical Association, commencing in the early twenties. Although living in an outer suburb (in the days of very bad roads), Croll was a most regular attendant at meetings of the Council and Branch, and rendered excellent service on many sub-committees, notably lodge, public health, building, obstetrical and publicity. He was, too, an excellent treasurer, and was responsible for the formation of the Medical Agency and the Finance Company.

He was at his best in the work of these committees. In large gatherings some members thought him taciturn and uncompromising and without humour. This was a great mistake, as his "taciturnity" was due to absorption in some matter on hand, and he had a good sense of humour, and rather enjoyed (in a good humoured way) taking a rise out of authority when authority became too pompous, as is sometimes the case.

When he took a matter up, either alone or in association with others, he certainly pursued the subject wholeheartedly. There were no half-measures, and he unrelentingly pursued a matter to the end—not only with keenness, but with enthusiasm.

He was an excellent man to follow in the hurly-burly of medical politics, and an excellent "second" whether the matter at issue was being settled on the home ground or with some outside body, such as a friendly society, local authority or government.

Croll was equally effective in the more sedate atmosphere of the Federal Council, and was an able representative of the Queensland Branch—at no time more so than in the fight he put up when it was proposed to restrict severely the activities of the Federal Health Department in tropical hygiene during the depression.

Others have written of Croll's scientific work, and I should like to emphasize from experience that, while not neglecting any new methods, he excelled in the older disciplines of medicine, notably that of observation.

Although connected for so many years with the Children's Hospital, Croll had links, through his wife, with the Brisbane Hospital in the days when his father-in-law, Mr. A. P. Payne, and Mr. Douglas Brown, the hospital's secretary and dispenser, respectively, were notable figures on the staff of the hospital.

Croll's example should be a spur to the oncoming medical men, and will be a source of solace to Mrs. Croll in her great loss.

JOHN JAMES SAVAGE.

WE regret to announce the death of Dr. John James Savage, which occurred on July 14, 1948, at Narrogin, Western Australia.

JAMES SPRENT.

WE regret to announce the death of Dr. James Sprent, which occurred on July 21, 1948, at Hobart.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

McDonald, John David, M.B., B.S., 1947 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Davies, George Bernard, M.B., B.S., 1947 (Univ. Sydney), c.o. Clifton Penny and J. R. Davies, Templecourt, corner of King and Elizabeth Streets, City.

McAuliffe, Peter Cornelius, M.B., B.S., 1947 (Univ. Sydney), 63, Telegraph Road, Pymble.

Hanbury, Paul Herbert, M.B., B.S., 1947 (Univ. Sydney), 172, Longfield Street, Cabramatta.

Budge, Alexander George Campbell, M.B., 1935 (Univ. Sydney), 15, Wentworth Street, Point Piper.

The undermentioned have been elected as honorary associates of the New South Wales Branch of the British Medical Association:

Brown, Kenneth James, 17, Dennis Street, Lakemba.

Broadfoot, Eric Murray, 569, Malabar Road, Maroubra Bay.

Cotton, Brian D., 6, Lisgor Road, Hornsby.

Green, Betty F., 10, Kimpton Street, Banksia.

Lindsell, Charles Kenneth, 18, Walenore Avenue, Kingsford.

Marnie, Philip Muir, 74, Snape Street, Kingsford.

Medical Appointments.

Dr. L. W. Jeffries, Dr. P. S. Messent, Dr. E. A. Johnson, Sir A. M. Cudmore and Dr. H. H. E. Russell have been appointed members of the Medical Board of South Australia.

The undermentioned appointments have been made at the Royal Adelaide Hospital, Adelaide: Honorary Ophthalmologists, Dr. G. H. B. Black, Dr. A. L. Tostevin; Honorary Assistant Ophthalmologists, Dr. T. L. McLarty, Dr. S. Pearlman, Dr. J. A. Rolland; Honorary Clinical Assistants, Dr. D. O. Crompton, Dr. Alfreda W. Thrush, Dr. I. G. Pavy; Honorary Surgeon, Dr. P. S. Messent; Honorary Gynæcologist, Dr. B. H. Swift; Honorary Dermatologist, Dr. L. W. Linn; Honorary Clinical Physiologist, Professor Sir Cedric S. Hicks; Honorary Anæsthetist, Dr. A. D. Lamphee; Honorary Assistant Anæsthetist, Dr. J. R. Cornish; Honorary Assistant Pathologist, Dr. J. M. Dwyer.

Diary for the Month.

AUG. 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
AUG. 4.—Victorian Branch, B.M.A.: Branch Meeting.
AUG. 4.—Western Australian Branch, B.M.A.: Council Meeting.
AUG. 5.—South Australian Branch, B.M.A.: Council Meeting.
AUG. 6.—Queensland Branch, B.M.A.: Branch Meeting.
AUG. 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute; Brisbane City Council (Medical Officer of Health). Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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